

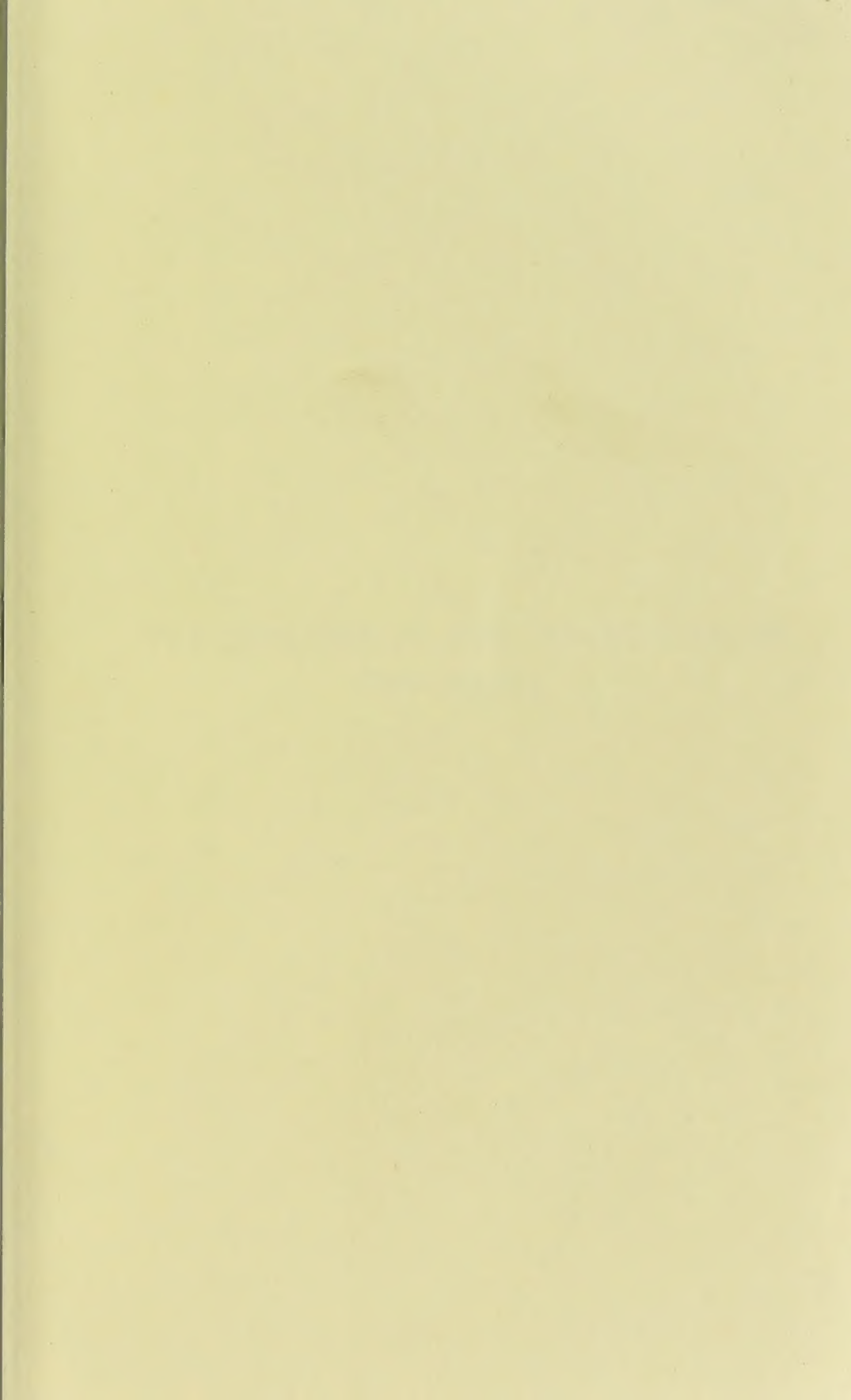






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June, 1917.

THE DISEASES OF THE HEART AND OF  
THE AORTA.





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THE  
DISEASES OF THE HEART  
AND OF  
THE AORTA.

BY  
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OF MILAN; ETC., ETC.

*Illustrated.*

"Vaga enim experientia, et se tantum sequens, mera palpatio est. . . . At cum experientia lege certa procedet, seriatim et continenter, de scientiis aliquid melius sperari poterit."—*Novum Organum*.

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TO  
WILLIAM STOKES, M.D., F.R.S., D.C.L. OXON.,  
LL.D. EDIN. AND CANTAB.;

REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF DUBLIN;

PHYSICIAN TO THE QUEEN;

PRESIDENT OF THE ROYAL IRISH ACADEMY; ETC., ETC.

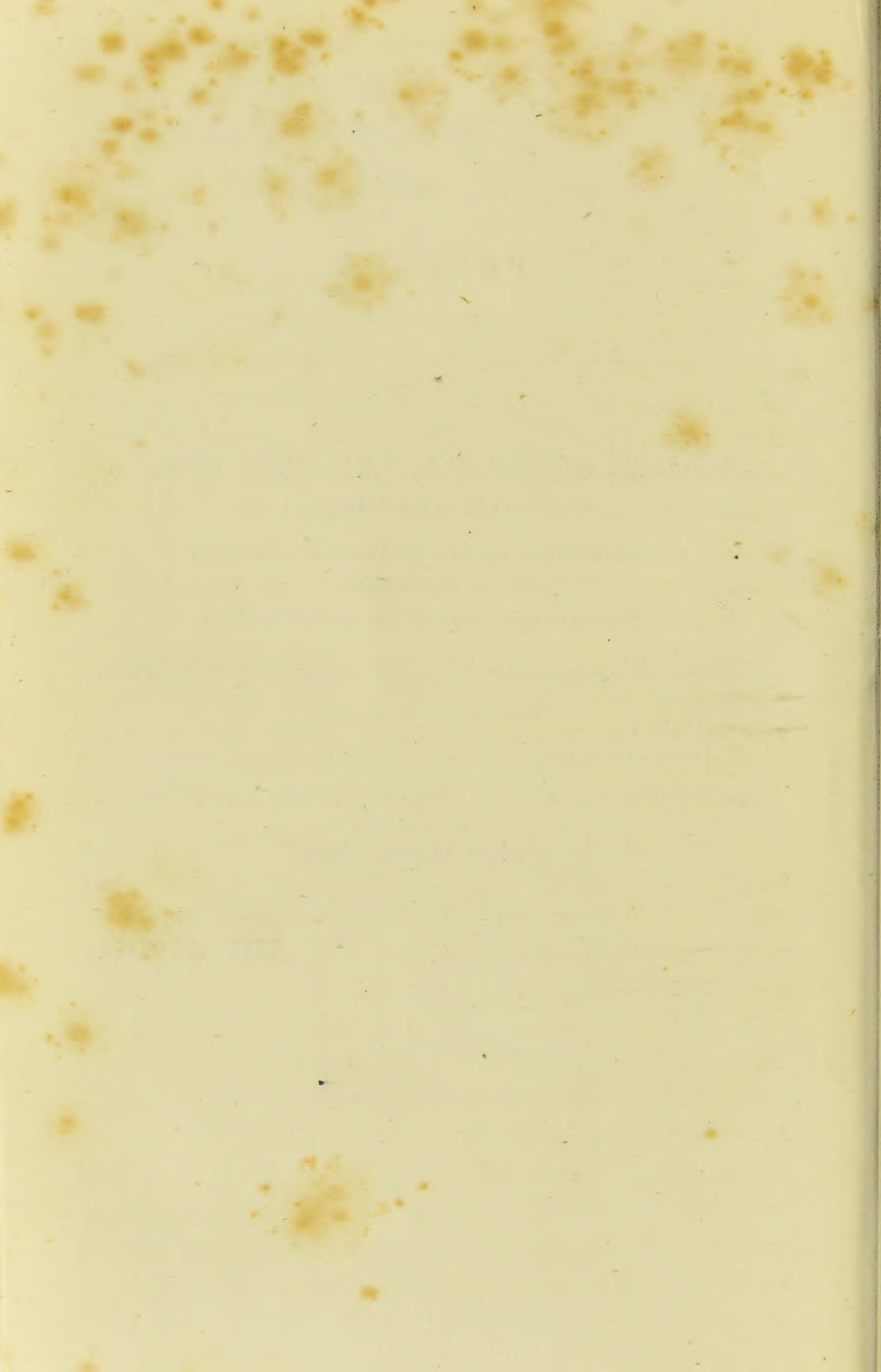
AS A TRIBUTE OF ADMIRATION FOR HIS EXALTED CHARACTER AND PRE-EMINENT  
ATTAINMENTS IN MEDICINE,

AND AS A TOKEN OF GRATITUDE FOR THE MANY AND GREAT FAVOURS  
RECEIVED FROM HIM THROUGH A PERIOD OF MORE THAN TWENTY YEARS,

THIS BOOK IS INSCRIBED,

BY HIS FRIEND AND FORMER PUPIL,

THE AUTHOR.





## P R E F A C E .

---

AT the outset it may be asked, why add another to the long list of treatises on the Diseases of the Heart already extant? To this question I would answer, firstly, because I think the subject needs revision in the light of modern pathology ; and secondly, because I believe that, as the result of long and earnest clinical study, I can contribute something towards placing this department of medicine upon a broader and firmer basis than that which it has hitherto occupied.

In undertaking a task of such magnitude, I was sensible of the great and numerous intrinsic difficulties which it involved, and no less conscious of the many defects inherent in myself, by which these difficulties were in no small degree enhanced. And now that the work, sketched in outline nine years ago, lies before me with all its imperfections, I am the less satisfied with my performance, because of the ideal standard of excellence at which I then aimed. For all such, I would solicit the considerate indulgence of my professional brethren ; especially of the Dublin School, to which I have the high honour to belong.

To the great masters, Corvisart, Laennec, Bertin, Bouillaud, Hope, Walshe, Stokes, and Corrigan, students of Cardiac Disease owe much. An acknowledgment is likewise due to many others, by whose labours our knowledge of this subject has been advanced. These eminent observers have not only laid the foundation of accurate knowledge in the domain of cardiac pathology, and constructed its rudiments out of the crude materials strewn around them by the labours of their predecessors, but have de-

veloped into maturity the infant science which they created. Despite the speculative physiology of the period, and the paucity of instrumental appliances then available for the purposes of physical exploration, in less than a quarter of a century from the date of Laennec's great work on *Mediate Auscultation*, the diagnosis of cardiac disease had attained to the proportions of a science. The present advanced state of knowledge of the physiology of the heart, due to the recent experimental researches of Chauveau and Faivre, E. Weber, Ludwig, the brothers Cyon, and, especially, those of Marey and his followers, has prepared the way for a more precise cardiac pathology. In no department of medicine is a full and exact knowledge of the normal function of the organs under examination of greater importance, than in that which embraces the study of Diseases of the Heart and Great Vessels. Without such knowledge, a rational system of diagnosis and treatment is impossible.

If an example were demanded of the direct application of physiology to the study of disease, no more forcible illustration of such could, in my opinion, be adduced, than that supplied by the heart. The entire cycle of cardiac phenomena in the state of health must be thoroughly apprehended, else a correct interpretation of the derangements exhibited in the various forms of disease to which the heart is subject, cannot be attained; and this, not only in regard to the rhythm and duration of these phenomena, but also in relation to their causes and associations, whether simple or multiple. Up to a very recent period, the knowledge available in this department of physiology was inadequate for the purpose of accurate clinical research; hence, the want of precision in regard to differential diagnosis which characterizes the works of many writers on Diseases of the Heart. The great mass of well observed facts, however, which they have collected; the correct, though broad lines of distinction they have drawn between the different genera

of Cardiac Disease ; the extensive knowledge of etiology and prevention, and the prescience in regard to results, shadowed forth in their writings, have contributed to the advancement of this important subject, in a degree scarcely appreciable by those who now engage in the same line of study.

It is no disparagement, therefore, to the labours of these distinguished men, and no slight upon their eminent services in the cause of medical science, to assert that their works require revision in order to bring them up to the standard of present knowledge. The recent edition (the fourth) of Dr. Walshe's admirable treatise, issued while this work was in progress, contains much of the information which it had been my ambition to supply. I venture to hope, nevertheless, that in so extensive a field, my labours may not be entirely unproductive of useful results.

This volume has far exceeded the limits originally designed. If an apology be needed, I can only plead, that a curtailment of its proportions, while it would have spared me much labour, would have been at the expense of an inadequate treatment of the subject.

As physician to a great hospital for the last thirteen years, I have enjoyed many advantages for the prosecution of my favourite study. The liberal and enlightened spirit in which the Mater Misericordiæ Hospital has been founded and conducted by the excellent Sisters of Mercy ; the admirable economic and general arrangements of the Institution ; and the unfettered liberty to admit patients conceded to the Medical Officers, combine to insure a supply of cases amply illustrative of any disease, to which, for the time, special attention may be directed.

For assistance rendered me in the preparation of this work, I am indebted to numerous kind friends. To the several gentlemen, now dispersed through many countries or settled in prac-



tice in their own, who acted as my clinical clerks, and assisted me in observing and noting the cases recorded in the text, and in applying the crucial test of diagnosis in the *post mortem* room, this book may, perhaps, serve as a not unpleasing memorial of our conjoint labour and common study. To my hospital colleagues, for the privilege, so kindly granted me, of free access to the patients under their charge; and, in an especial manner, to Dr. Nixon, for the many admirable sphygmograms dispersed through the succeeding pages with which he has supplied me, I beg to express my deep sense of obligation. For the great and valuable assistance rendered by my friend Dr. George F. Duffey, in revising the sheets (from page 225), and pointing out references, and especially for the preparation of the copious Index, which has been entirely executed by him, I hereby tender my warm and sincere thanks. The woodcuts have been executed by Mr. George A. Hanlon.

30, Harcourt-street, Dublin.

July, 1875.

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### CORRIGENDA.

- Page 60, last line, *for* Archiv., *read* Archiv.  
,, 70, line 29, *for* diagramatic, *read* diagrammatic.  
,, 78, ,, 3, *for* dilitation, *read* dilatation.  
,, 91, ,, 25, *for* Chaveau, *read* Chauveau.  
,, 96, last line, *for* Hospitaux, *read* Hôpitaux.  
,, 106, ,, 31, *for* diagonosis, *read* diagnosis.  
,, 108, ,, 2, *for* æriform, *read* aeriform.  
,, 112, ,, 17, *for* diognosis, *read* diagnosis.  
,, 115, ,, 37, *for* shoc, *read* shock.  
,, 123, ,, 4, *for* interrupted, *read* continuous,  
,, 129, 162, and 163, *for* Guttemann, *read* Guttmann.  
,, 129, ,, 16, *for* Giegel, *read* Geigel.  
,, 145, ,, 38, *for* shoc, *read* shock.  
,, 739, ,, 17, *for* cysticeri, *read* cysticerci.  
,, 804, ,, 27, *for* sitting, *read* recumbent.  
,, 842, ,, 24, *for* M. Guizot, *read* M. Guyot.

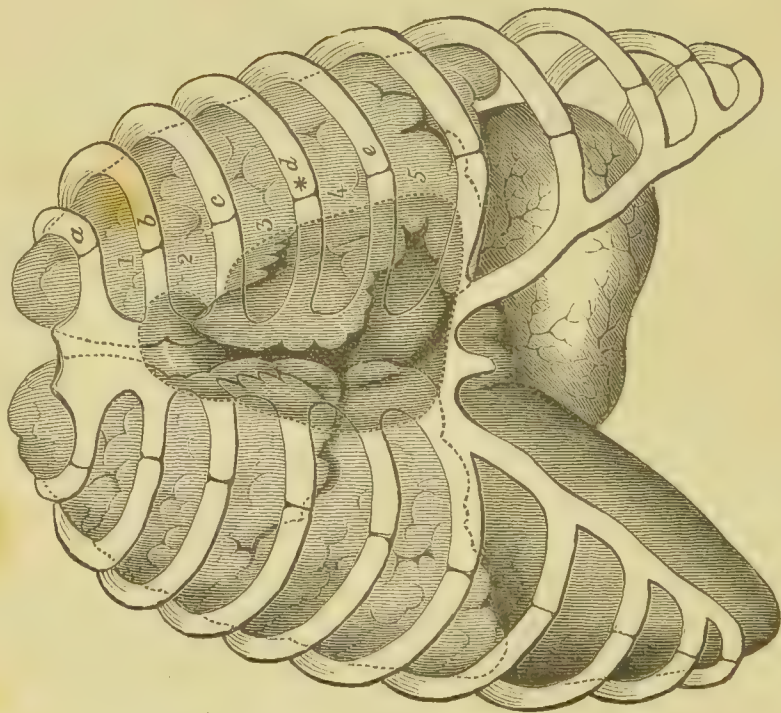


FIG. A.

Showing the relative position of the heart, lungs, stomach, and liver: also the outline and extent of the regions of superficial and deep precordial dulness.

The letters indicate the ribs and costal cartilages; the numerals indicate the corresponding intercostal spaces; the asterisk indicates the left nipple. For description see page 1. The figures have been modified from Flint.

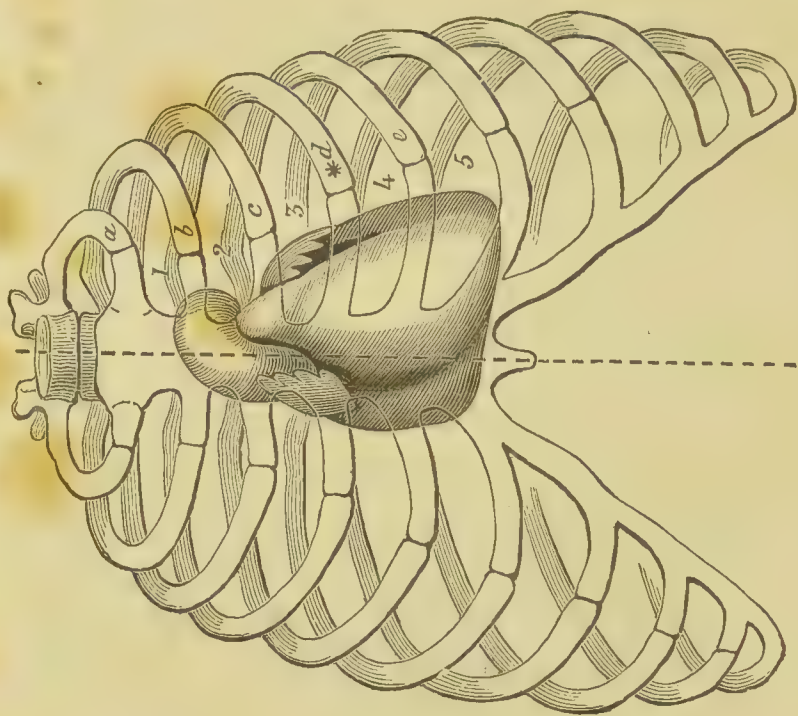


FIG. B.

Showing the outline of the heart, and the position of its several parts in relation to the walls of the chest and the nipple.

The asterisk indicates the left nipple. For description see page 1. The figures have been modified from Flint.

# THE DISEASES OF THE HEART AND THE AORTA.

---

## CHAPTER I.

### ANATOMY AND PHYSIOLOGY OF THE HEART.

THE heart, the centre of the circulatory system, occupies the lower part of the mediastinum, and the anterior inferior and internal portion of the left side of the thorax; it is enclosed in a twofold membranous sac, the pericardium, and interposed between the pleuræ, which, in close juxta-position by their outer surfaces anteriorly and posteriorly, forming the lateral boundaries of the corresponding portions of the mediastinum, are divaricated in the intermediate space to receive the heart and pericardium.

The region in front of the heart is named the *precordium*, and that portion of it in which the pericardium is not covered by the edges of the lungs, the *superficial precordial region*. This last-mentioned space, in which the pericardium comes into contact with the anterior wall of the chest, is quadrilateral in figure as correctly described by Sibson.\* It measures about three inches in its transverse, and two inches in its vertical diameter; bounded superiorly by a horizontal line corresponding to the upper edge of the fifth costal cartilage, and extending from the middle line of the sternum to a point within an inch and a-half of the left nipple line; below, by a line on a level with the upper edge of the sixth costal cartilages; on the right side, by the mid line of the sternum; and on the left, by a curved line, convex outwards, commencing one and a-half inch inside the nipple line at the upper edge of the fifth costal cartilage, and terminating at the junction of the sixth and seventh costal cartilages near the sternum, two inches internal to the nipple line. These boundary lines are constituted on three sides by the edges of the lungs, viz., on the right side by the edge of the right lung, and superiorly and

\* *Medical Anatomy.*



externally by that of the left lung. The inferior boundary is formed by the diaphragm. This space corresponds to the area of absolute precordial dullness, but varies much in figure and dimensions in different individuals, being less extensive in the robust, and occasionally all but abolished in the subjects of pulmonary emphysema. The dimensions given are those presented at the acme of inspiration; but during expiration they are considerably extended, owing to retrocession of the edges of the lungs.

Gendrin \* says the position of the edge of the left lung may be marked out by a line drawn from the junction of the cartilage of the second rib with the sternum, downwards and outwards to the extremity of the last rib; and that of the right lung by a line carried from the upper extremity of the mesial line of the sternum, downwards and outwards to the anterior extremity of the first right false rib. In the dead body they do not advance so far as in the living.

The space known as the *deep precordial region*, or that within which modified cardiac dulness is detectible during life, corresponds in figure and dimensions to the anterior surface of the heart. It is irregularly triangular in outline, the vertex above formed by the transverse portion of the arch of the aorta, and extending from the upper edge of the second costal cartilage of the left side at its junction with the sternum, to the lower edge of the corresponding portion of that of the right side. The vertex is rounded, and about two and a-half inches in diameter. The base is below, and corresponds to a horizontal line about four inches in length, connecting the lower edges of the sixth costal cartilages of the opposite sides. The left boundary, slightly convex outwards, extends between the upper edge of the second costal cartilage of that side, half an inch external to its union with the sternum, and the upper edge of the sixth of the same side, one inch inside the nipple line. The right boundary corresponds approximately to a line carried from the lower extremity of the right second chondro-sternal articulation, vertically downwards to the upper extremity of that of the sixth. This region includes within its boundaries that known as the superficial precordial region, and yields, to the extent of the latter, *absolute* percussion dullness, but elsewhere *qualified* and barely perceptible

\* *Leçons sur les Maladies du Cœur et des Grosses Artères*, tom. i., 1841-42.

dullness, which, when the edges of the lungs are inflated, is converted into absolute resonance.

The pericardium consists of a fibrous and serous layer, the former, external, conical in figure, the base resting on the central leaflet of the cordiform tendon of the diaphragm, with which in the adult it is intimately incorporated, presents nine funnel-shaped openings for the passage of the great vessels which enter and leave the heart, viz., the aorta, the two primary branches of the pulmonary artery, the superior and the inferior vena cava, and the four pulmonary veins. In the foetus there is an additional aperture for the ductus arteriosus, but after the closure and atrophy of this vessel, that opening becomes likewise obliterated. The edges of these several openings are everted, and incorporated with the outer coats of the vessels, with the single exception of the ascending vena cava, which pierces the diaphragm and the fibrous pericardium sharply, and at once enters the right auricle. This outer layer of the pericardium consists of fibrous tissue, chiefly of the white or inelastic kind, the fasciculi of which mutually intersect at all conceivable angles; there is likewise a large proportion of yellow elastic tissue, consisting of curling and branching filaments, varying in diameter from 1-10,000th to 1-6,000th part of an inch, and unaffected by strong acetic acid.

The serous layer of the pericardium, after lining the fibrous envelope, is reflected thence upon the great vessels at the base, to the heart, which it completely invests, constituting the *epicardium*; it forms a common envelope for the aorta and the pulmonary artery, about two inches of which are within the pericardium; the portions of the vessels to this extent, which are in mutual contact, being alone uncovered by serous membrane. From the root of the aorta and pulmonary artery the serous envelope is reflected upwards over the anterior and superior surfaces of both auricles, to the bifurcation of the pulmonary artery, whence it is reflected forwards upon the trunk of that vessel, and by it conducted downwards to the ventricles, expanding in its course into the common envelope for the aorta and pulmonary artery, already described. To the right it is reflected over the descending cava, covering it on the anterior half of its circumference to the extent of one inch of its length; it thence

passes downwards in front of the right pulmonary veins, to the ascending vena cava, which, to the extent of half an inch of its length, it invests anteriorly and laterally. Between the two cavæ, and in front of the right pulmonary veins, the serous membrane dips into an oblong fossa, the long diameter of which is vertical, and the depth of which is increased by tension of the venæ cavæ. From the roots of the great vessels at the base it descends over the anterior surface of the heart, to the inferior margin and apex, whence it is reflected over the inferior surface, and upwards over the posterior surface, investing in the last-mentioned portion of its course the posterior surface of the left ventricle and auricle; and from the upper margin of the latter, ascends to the bifurcation of the pulmonary artery, where, on a level with the anterior reflection previously described, it is reflected backwards to the posterior wall of the pericardium, and thus descends to the diaphragm. On the right and left side it invests the posterior surface of the pulmonary veins at their termination, to the extent of one inch of their length, being also inflected forwards between them to a variable extent. In some subjects this inflection does not exist. The lower surface of the inferior pulmonary vein, on both sides, is completely invested by serous membrane, whilst the superior surface is, to an equal extent, uncovered. The anterior surfaces of these vessels are covered only to the extent of about half an inch.

It follows, therefore, that the sac of the pericardium is divided by the reflection of its serous lining into two great pouches or recesses, viz. : one, by much the larger, in front of the heart, and separated at its circumference from the other by a double septum, formed by the reflection, *dos à dos*, of the two layers of serous membrane already described. This septum, on the right and left sides, includes the corresponding pulmonary veins, whilst superiorly its constituent laminae, extended somewhat tensely between the superior posterior margin of the auricles inferiorly, and the bifurcation and both lateral branches of the pulmonary artery superiorly, are separated only by some dense areolar tissue. The anterior recess is traversed superiorly by the trunks of the aorta and pulmonary artery, which, through the greater portion of their course within the pericardium, occupy a common



tubular passage between the two layers of serous membrane, but near their point of exit from it acquire each a proper serous investment.

The venæ cavæ, and both branches of the pulmonary artery, may be regarded as outside the pericardium; projecting, however, to an unequal extent of their circumference into its cavity, the former in the posterior, and the latter in the superior wall.

The subserous lamina is of variable thickness and density in different portions of its extent; thus, beneath the fibrous pericardium it is represented by the deepest portion of that membrane, which is somewhat less dense in structure than the remainder; in this stratum the minute vessels and capillaries of the pericardium are distributed; on the great vessels it is more lax, admitting of easy detachment of the serous layer; whilst upon the heart it forms a remarkably dense but thin lamina, the *fascia cordis*, which is inseparably united with the basement membrane and epithelium, upon the one hand, and with the sparse perimysium or connective tissue of the heart, upon the other. In the course of the cardiac grooves only, where the principal blood vessels and nerves with a variable quantity of fat are lodged in its meshes, is this tissue sufficiently lax to admit of easy separation of the serous investment from the heart. In the interspaces between the grooves and over the surface of the heart generally, imbedded in the subserous lamina, the lymphatic vessels and the primary branches of distribution of the cardiac nerves are spread out. This tissue is likewise the seat of the "white," or "milk-spots." There is usually but one such, which is situated upon the anterior surface of the right ventricle, irregularly circular, and varying in size from a four-penny to a half-crown piece. When additional spots are present, they are found upon the anterior surface of the apex and that of the right auricular appendix. These latter are never met with in the absence of the former, which, on the contrary, exist in greater or less perfection on the hearts of most adult and aged persons, unaccompanied by similar appearances on other portions of the surface of the heart. Such spots were formerly, but incorrectly, regarded as the result of limited pericarditis. I have repeatedly examined them with much care, but never found

fibrinous exudation upon the epithelial surface, which here, as upon other portions of the surface of the heart, has invariably, in my experience, presented the polished and glistening appearances characteristic of its healthy condition. They arise, manifestly, from chronic irritation of the superficial surface of the heart, the result of excessive and long continued friction, to whatever cause due. Persons long engaged in some bodily labour, involving great muscular strain and habitual distention of the right chambers of the heart, are most likely to exhibit typical "milk spots." Hence, their usual position on the anterior surface of the right side of the heart, opposite the unyielding surface presented by the sternum.

Examined microscopically, the subserous lamina of the pericardium is found to consist mainly of white fibrous tissue, the fasciculi of which overlie and interlace with one another at all conceivable angles. It includes, also, a large proportion of yellow elastic tissue, composed of fine curling filaments of an average diameter of 1-17,000th part of an inch. This latter tissue confers upon the pericardium in an eminent degree the property of elasticity, in virtue of which it is capable of accommodating itself to variations in volume of the several chambers of the heart, during contraction and relaxation respectively. The capacity of the pericardium is accurately adapted to the volume of the heart at all periods of cardiac action. Its walls are therefore constantly in a state of tension during the continuance of cardiac movement, no less during the systole than the diastole of the ventricles.

This accuracy of adaptation of the pericardium is, no doubt, mainly due to the constancy of volume of the heart at all periods of its action, a circumstance to be attributed to the alternating contraction and relative capacity of the auricles and ventricles, and of the roots of the great vessels connected with them, and included within the pericardium. If the chest of a still living animal be laid open, it will be found impossible to pinch up the pericardium between the finger and thumb, and difficult even by means of a pair of good forceps, as long as the heart continues to pulsate.

The purposes of the pericardium seem to be to insulate the

heart, and by presenting to it a smooth and moist surface, and excluding adjacent organs, to insure its free and unimpeded movement in the performance of its function.\*

There is yet another purpose served by the pericardium, to which attention has not been hitherto directed, namely, that of protecting the heart from mediate atmospheric pressure at these periods of its action, when, owing to asynchronism between its movements and those of the lungs and thorax, the circulation would be liable to obstruction in the more thin-walled of its chambers, and in the roots of the great vessels, from pressure of the expanding lungs or receding chest walls.

The pericardium is firmly attached, in front, to the sternum, through the anterior mediastinum; posteriorly, to the vertebral column through the posterior mediastinum; superiorly, to the first ribs and clavicles through the reflections of the cervical fascia upon the great vessels connected with the aorta and descending cava, which are themselves incorporated by their external coats with the fibrous lamina of the pericardium where they respectively penetrate it, and with the serous layer where the latter is reflected upon them. The phrenic nerves likewise connect the pericardium superiorly with the cervical vertebræ, and their arterial *comites* with the subclavian arteries through the medium of the internal mammaries.

Inferiorly the pericardium is fixed and held tense by its attachment to the great phrenic centre of the diaphragm; and by the tension of the pleura reflected from it upon the lung on either side, it is subjected to constant, considerable, and in the normal state, equal traction laterally.

It is the derangement of balance between the tractile forces thus exercised upon the two sides of the pericardium, owing to collapse of either lung, which determines its lateral displacement to the opposite side in cases of copious aeriform

\* Senac (*Traite de la Structure du Cœur, de son Action, et de ses Maladies*, 1749) says the uses of the pericardium are to facilitate the movements of the heart, by presenting opposed moist surfaces, the halitus being confined and thereby preserved, and to fix the roots of the great vessels, and maintain unalterable relationship between them and the heart, and thus favour the entrance and exit of the blood.

or liquid effusion into one side of the chest, according to the experiments of Dr. R. Douglas Powell,\* to which I shall have occasion to revert.

Between these several fixed points of attachment the pericardium is kept in a state of tension sufficient to neutralize the pressure of the expanding lungs, and to diminish, at least, that of the receding sternum. Were it otherwise, the blood would be inevitably forced back from the auricles upon the great veins, as often as auricular diastole should happen to coincide with the acme of inspiration, during which the anterior edges of the lungs advance between the pericardium and the sternum with a force equivalent to several inches of mercury; and a like inconvenience, but in a much less degree, would probably arise from the retrocession of the sternum at the acme of expiration, were it not for the protection afforded to the subjacent auricles by the tense intervening wall of the pericardium.

From what has preceded, it follows that the pericardium is endowed, in an eminent degree, with the property of resistance to extension. To this property, conjoined with the strict adaptation of its capacity to the volume of the heart, must be attributed the formidable train of symptoms, of a syncopal character, which follows from rapid effusion, of whatever kind, into the sac of the pericardium.

In such a case the effused liquid is interposed between a tense and unyielding membrane, and an organ whose chambers alternately contract and expand. Now, inasmuch as the force of expansion of any of the chambers of the heart is the equivalent of the blood-pressure expended upon its internal surface during its relaxation, it is manifest that this pressure must be sufficient, not only to divaricate the walls upon which it is exercised, but likewise to overcome the resistance of the pericardium, else the expansion of the chamber will be limited by the effusion, and the volume of blood circulating through it diminished in the same proportion. Hence, the feeble impulse of the heart, the weak and flickering pulse, and the pallid and congested surface in cases of moderate but acute effusion into the pericardium, and

\* *Medical Times and Gazette*, 30th January, 21st August, 1869, and *British Medical Journal*, 17th July, 1869.



the mortal syncope that not unfrequently ensues when the effusion has been rapid and copious.

Sibson says that twelve to eighteen ounces of liquid may be injected into the healthy pericardium. This result is due, in part, to forced expansion of the sac, but mainly to evacuation of the chambers of the heart. In chronic pericarditis as much as three pints of serous effusion have been found in the pericardium, the structure of the sac having undergone slow and general extension.

If the pericardium be laid open whilst the body is still warm, its serous surface will be found moistened throughout by a fine halitus, which, coincidently with the cooling of the body, becomes condensed into a clear serum, amounting in quantity to a few minims. The surface whence this moisture is exhaled is smooth, glistening, and transparent, and coated with a single layer of nucleated pavement epithelium, the particles of which are oval in shape, and of an average long diameter of 1-1100th part of an inch, and a short diameter of 1-1700th.

This surface, when the subject of inflammation, loses its smoothness and transparency, and becomes studded with effused lymph, affecting a flocculent or reticular disposition, whence arises the phenomenon of *frottement* or friction-sound during the movements of the heart. The heart is now likewise brought into view, the portions of the organ visible from the front being the greater part of the right ventricle, a part of the left ventricle including the apex, the appendices of both auricles, and a part also of the sinus of the right auricle. The intra-pericardial portions of the pulmonary artery, aorta, and superior vena cava are likewise exposed to view, as are the trunks and primary branches of the two coronary arteries occupying the anterior cardiac grooves.

The heart is irregularly triangular, and furnishes by its figure a standard of comparison, bodies shaped like it being described as *cordiform*. It presents three surfaces, of which the posterior is convex, resting superiorly on the posterior mediastinum and in immediate contact with the oesophagus, and inferiorly in a deep fossa on the internal surface of the left lung. This surface is formed entirely by the left auricle and ventricle, and presents the auriculo-ventricular groove, containing a branch of the ante-

rior coronary artery, a branch of the coronary vein, and numerous branches of the anterior coronary plexus of nerves. The inferior surface is flat and horizontal, formed in five-sixths of its extent by the ventricles, and in the remaining sixth by the right auricle; of the ventricular portion of this surface the anterior third is formed by the right ventricle, and the posterior two-thirds by the left ventricle. It presents the continuation of the ariculo-ventricular, and the so-called posterior interventricular groove; the former containing the continued trunk of the posterior coronary artery, and an anastomosing branch from the anterior coronary, together with branches of the cardiac nerves, and the great coronary vein; and the latter, the terminal branch of the posterior coronary artery, branches of the cardiac nerves, and a large tributary of the coronary vein. This surface rests upon the central leaflet of the cordiform tendon of the diaphragm, and to a slight extent upon the muscular portion between the central and left leaflets. The anterior surface of the heart, irregularly triangular in outline, and slightly convex, presents to view the anterior interventricular, and the auriculo-ventricular grooves, in which are lodged the coronary arteries and nerves; likewise the roots of the pulmonary artery and aorta overlapped by the auricular appendices. It is formed in the basic sixth of its extent by the auricles, chiefly the right, and in five-sixths by the ventricles, the right ventricle constituting two-thirds, and the left only one third of this latter portion. It is related, in the superficial precordial region, to the lower portion of the sternum left of the middle line, the left costal cartilages from the middle of the fourth to the upper margin of the sixth, and the corresponding intercostal spaces to one inch inside the nipple, and to the left triangularis sterni muscle and internal mammary vessels, with the intervention only of the pericardium and some lax areolar and adipose tissue. The dimensions of this region, which corresponds to that of absolute precordial dulness, vary not only in disease, but likewise in health; thus, whilst the lungs recede in expiration it is considerably extended, and in healthy persons with well-developed lungs it may be much contracted. In hypertrophy of the heart unassociated with pulmonary emphysema it may be greatly enlarged; whilst, in the last-mentioned disease, it is always much

reduced, and may be even entirely abolished. In the remainder of its extent the anterior surface of the heart is covered by the lungs. Its general outline, and the situation of its principal parts, may be conveniently indicated by the following lines and fixed points on the anterior wall of the chest. The superior boundary line of the auricles, and the roots of the great vessels connected with the base of the heart, correspond to the upper edge of the third costal cartilages. The right extremity of the base, formed by the right auricle, is indicated by a curved line, convex to the right, commencing a little to the right of the mid sternal line on a level with the upper edge of the third costal cartilages, thence passing downwards and to the right, crossing the third intercostal space and fourth costal cartilage half an inch from the sternum, and curving slightly inwards and downwards to terminate in the fifth interspace, somewhat less than half an inch to the right of the sternum. The inferior boundary of the heart may be determined by a line carried from the last-mentioned point towards the left side and slightly downwards, a quarter of an inch above the base of the xyphoid cartilage, crossing the common cartilage of the sixth and seventh ribs of the left side at its junction with the sternum, thence passing horizontally into the fifth intercostal space, to terminate in that space at an equal distance from the two adjacent ribs, beneath the junction of the fifth rib with its cartilage, two inches below the level of the nipple, and one inside the nipple line. The left boundary is mapped out by a slightly curved line, convex to the left side, commencing at the upper edge of the left third costal cartilage at the junction of its middle with its outer third, passing obliquely downwards and outwards, crossing the fourth costal cartilage half an inch inside its junction with the corresponding rib, and thence passing downwards, with a slight inclination inwards, to terminate in the fifth interspace, one inch to the right of the nipple line.

The line of the auriculo-ventricular sulcus, slightly curved, and convex to the right side, commences at the middle of the left third costal cartilage, and terminates at the junction of the sixth and seventh right common cartilage with the sternum.

The orifice of the pulmonary artery is beneath a point in the

second intercostal space, immediately to the left of the sternum, and that of the aorta, the left two-thirds of which are covered by the orifice of the pulmonary artery, is behind the lower portion of the left third chondro-sternal articulation, and the adjacent portions of the cartilage and sternum.

The centre of the left auriculo-ventricular orifice corresponds to a point on the left third costal cartilage, less than an inch from the sternum. This orifice, being remote from the surface of the chest, and covered by the great vessels, is unfavourably circumstanced for auscultation; the sounds having their origin in it are therefore most distinctly audible at the apex. In like manner, the orifice of the aorta being overlain in greater part by that of the pulmonary artery, sounds, whether normal or abnormal, originating in it, are most distinctly heard, and with the least liability to a mistake of identity, at the centre of the sternum on a level with the lower edge of the third costal cartilage, and thence obliquely upwards beneath the inner portion of the second right intercostal space and costal cartilage. The right auriculo-ventricular orifice is behind the centre of the sternum, at the level of the fourth intercostal space. The ascending portion of the arch of the aorta bulges a quarter of an inch beyond the right border of the sternum, and the vena cava superior projects half an inch outside the aorta in the first and second right intercostal spaces, and behind the second costal cartilage. [Quain and Sharpey]. The trunk of the pulmonary artery is entirely to the left of the mesian line, and its left border projects three-fourths of an inch beyond the left margin of the sternum. The right auricular appendix covers the root of the aorta to the right, and on a level with the orifice of the pulmonary artery, and its point is a little to the right of the middle line of the sternum on a level with the upper edge of the third costal cartilages. The *conus arteriosus* of the right ventricle extends from the lower extremity of the body of the sternum, obliquely upwards to the inner portion of the left second intercostal space. The apex of the left auricular appendix is one and a-quarter inch to the left of the sternum, and behind the third costal cartilage. The summit of the arch of the aorta is on a level with the middle of the manubrium sterni; and the left edge of the arteria innominata at its



origin is a little to the left of the middle line. The concavity of the aortic arch is on a level with the junction of the manubrium with the body of the sternum. [Allen Thompson.]

The axis of the heart corresponds to a line traversing the fifth intercostal space, three and a-half inches to the left of the middle line, passing obliquely upwards, backwards, and to the right, crossing at an acute angle, and in succession, the bodies of the eighth, seventh, and sixth dorsal vertebræ, and the corresponding intervertebral discs, and passing, if prolonged, through the centre of the right scapula. (*See Plate 1, Fig. i. and ii.*)

The heart is a quadrilocular organ, consisting of two auricles and two ventricles; functionally, however, it is double, being composed of a right and a left portion, which, normally, have no direct communication with one another after birth. During foetal life a direct passage exists from the right into the left auricle, known as the foramen ovale, or of Botal, and the heart is consequently a single organ at that period of life. Each side of the heart consists of an auricle and a ventricle, freely communicating by the auriculo-ventricular opening, by which a current is permitted from auricle to ventricle, but is intercepted in the opposite direction by a valvular apparatus. The right auricle receives, through the two venæ cavæ, the venous blood of the entire body, except that from the walls of the heart, which enters it by the great coronary vein and the venulæ Thebesii. The blood thence passes into the right ventricle, whence it is conveyed to the lungs by the pulmonary artery, and after undergoing aeration, or conversion into the arterial state in these organs by the respiratory process, it is returned to the left auricle by the four pulmonary veins, and passed into the left ventricle, whence it is conducted by the aorta and its branches to all parts of the body. Having subserved the functions of nutrition and secretion in the various parts of the organism, the blood is conducted back in a venous state to the right auricle of the heart.

Such is a general and very brief sketch of the circulation, as discovered and demonstrated in the seventeenth century by the illustrious Harvey.

The right chambers of the heart are likewise anterior to those of the left side. The auricles constitute the base, and the ventricles the apex and principal portion of the heart.

*Interior of the Heart.*

The auricles are most conveniently laid open for examination by a crucial incision made into the external anterior surface of the right, and the external posterior surface of the left. Each is divisible into two portions, a *sinus* and an *appendix*, and lined throughout by a fine membrane, continuous with that covering the inner surfaces of the blood vessels, and named endocardium by Bouillaud, in contradistinction to the outer or investing serous membrane, the *exocardium*. This membrane belongs structurally to the serous class, being composed of two layers, viz.: on the inner or free surface an epithelial stratum, consisting of a single layer of flat, oval, nucleated, and tessellated particles, having an average long diameter of 1-1700 and a short diameter of 1-3500 part of an inch in the foetus of six months. These are supported externally by a layer of fine connective tissue, in which the elastic element preponderates in the auricles. The auricular sinuses are smooth throughout, with the exception of a portion of the anterior wall of the right, but the appendix of each presents an uneven or reticulated surface, owing to the presence upon it of a great number of fleshy columnar projections, or pilasters, which, with a few connecting bands, are disposed in parallel lines from the apex towards the base of the appendix, and thence, in the right auricle, downwards upon the anterior wall of the sinus, as far as the auriculo-ventricular opening. In the left auricle these muscular projections are confined to the appendix, in which they are of greater magnitude than those of the right side.

The *right auricle* forms the anterior and inferior portion of the base of the heart. The appendix projects from its left and superior portion: it is irregularly conical, dentated at the margins, and curved, embracing the root of the aorta on the right side, and overlapping it somewhat by its apex.

In capacity the right auricle is generally considered somewhat to exceed the left. Its sinus is cuboid in figure, and presents on the superior wall the orifice of the descending vena cava to the right, and the narrow passage of communication with the appendix to the left side. The former is ovoid in figure, not protected by a valve, and its axis tends downwards, inwards, and somewhat forwards, in the direction of the auriculo-ventricular

opening. On the inferior wall of the sinus are seen three apertures, viz.: the auriculo-ventricular, that of the coronary sinus, and that of the ascending vena cava, placed from before backwards, and from left to right, in the order in which they have been just enumerated.

The auriculo-ventricular orifice is nearly circular in figure, and about one and a-quarter inch in diameter; it is bounded by a tendinous ring, smooth and even on the inner surface, and leads directly into the right ventricle. The opening of the coronary sinus is about three lines in diameter, and guarded by a circular or bilunate valve composed of a duplicature of the endocardium only. External to this valve is the coronary sinus, into which the great coronary, and a few minor veins open, the former guarded by a double valve.

The orifice of the inferior cava is considerably larger than that of the superior vessel of the same name; it has a direction upwards and inwards, and is guarded by the Eustachian valve anteriorly. This valve is of considerable magnitude in the foetus, serving at that period of existence to conduct the blood from the inferior cava, through the *foramen ovale*, directly into the left auricle; but at birth, when the adult circulation has been established, and its special function thereby abolished, it commences to undergo progressive atrophy, and in the adult is usually represented only by a narrow crescentic fold of membrane.\*

The posterior and left wall of the auricle is formed by the septum auricularum. On this is situate a smooth prominence superiorly, intermediate between the orifices of the venæ cavæ, and named the *tubercle of Lower*. Still lower down, and immediately over the mouth of the inferior cava, a dimple or superficial depression may be seen. This is the *fossa ovalis*, the vestige of the foramen ovale already mentioned, by which, in foetal life, blood passed directly from the right to the left auricle; the closure of this passage, which occurs at birth, and signalizes the transition from the foetal to the adult state of existence, is not in all persons complete, and hence, occasionally, admixture of the venous with the arterial blood, giving rise to the condition known as *cyanosis*. The fossa ovalis is embraced in three-fourths of its circumference,

\* Senac (*opus citat*) has seen it entire as late as fifteen years after birth.

viz.: anteriorly, superiorly, and posteriorly, by a smooth, prominent, muscular crescent, the *annulus ovalis* of Vieussens. To the anterior cornu of this crescent the left extremity of the Eustachian valve is attached, an arrangement by which, in the foetus, the passage of blood from the inferior cava through the foramen ovale is insured.

Besides the openings already mentioned, a number of minute apertures are visible, dispersed over the smooth portion of the sinus; these are the *foramina Thebesii*, the majority of which are shallow recesses in the wall of the auricle, and lined by an inflection of the endocardium; a few, however, usually three or four in number, are the orifices of minute veins, the *venulæ Thebesii*, which open into the inferior anterior portion of the auricle. One of these is the *vein of Galen*, derived from the right border of the heart; another is that of the infundibulum of the right ventricle. M. De Lannelongue\* describes four of these veins opening into the right auricle upon the interauricular septum, but subject to slight variation in number and position. One is the vein of Galen, which opens into the appendix near its right wall; another, near the superior vena cava; a third, intermediately between the orifice of the superior cava and that of the auricular appendix; and a fourth, near the opening of the coronary sinus.

The mouths of all these vessels are funnel-shaped, and communicate freely with one another by large connecting channels beneath the lining membrane of the auricle, provided with valvular orifices. These large orifices he designates as "foramina;" but there are generally several others of much smaller size and similar conformation dispersed over the septum, and in the vicinity of the auriculo-ventricular opening, and communicating with a fine sub-surface venous plexus, which is occasionally connected with the former. These are the formina of Vieussens and of Thebesius, and distinguished from the former by the designation of "forminula."

The *left auricle*, situated superiorly, posteriorly, and at the left extremity of the base, is bounded in front by the roots of the

\* Circulation veineuse des parois auriculaires du cœur, quoted by A. Luton. *Nouveau Dictionnaire de Médecine et de Chirurgie Pratique*; Paris, 1868: article "Cœur."



aorta and pulmonary artery, the latter being overlapped on the left side by its appendix, which is somewhat longer and more curved than that of the right auricle; posteriorly, by the œsophagus, and more remotely, the thoracic aorta and other contents of the posterior mediastinum, through the medium of the pericardium; externally, by the left lung; internally and somewhat anteriorly, by the right auricle; inferiorly, by the left ventricle; and superiorly, by the left branch of the pulmonary artery, with the intervention of the pericardium. The sinus is throughout smooth on its internal surface, presenting no muscoli pectinati, which are restricted to the appendix, and of much greater development than those in the right auricle; at the four angles of its posterior wall, which is somewhat convex forwards, are situate the orifices of the pulmonary veins, two on the left and two on the right side, the former usually in closer proximity than the latter, and frequently communicating with the auricle by a common orifice. These orifices are ovoid in figure, have an oblique aspect forwards and inwards, and are unprovided with valves. Several minute foramina Thebesii are likewise dispersed over the surface of the sinus, and one of large size opens on the posterior wall, between the orifices of the pulmonary veins, and conducts venous blood from the glands at the bifurcation of the trachea.

The *right ventricle* forms the inferior, anterior, and right portion of the heart, not extending, however, quite to the apex; it is crescentic in section, or rather irregularly triangular, the anterior wall being more extensive than the inferior, whilst the posterior and left wall, which is convex towards the right ventricle, is the most extensive of its boundaries, and formed entirely by the septum ventriculorum. The cavity of the right ventricle consists of two portions, the ventricle proper, constituting its greater portion, and the sinus or infundibulum, a smooth funnel-shaped passage, leading from the anterior, superior, and left extremity of the former, into the pulmonary artery. The ventricle proper is broad towards the base, and narrow towards the apex of the heart, and throughout, except at the auriculo-ventricular opening, its internal surface is reticulated by round or angular fleshy columns, the *columnæ carneæ*, which intersect at various angles, and are of three kinds, viz.: a set which are attached to

the wall of the ventricle by one surface throughout their entire length, and present the appearance of pilasters; another, much more numerous, attached by their extremities and free in the intermediate portion; and a third, the *musculi papillares*, usually three in number, much larger than either of the former, conical in shape, connected with the ventricular wall by the larger extremity, and by the smaller end, which is mammillated and usually bifid, giving attachment to the *chordæ tendineæ*, and through these connected with the auriculo-ventricular valve. The *musculi papillares* arise towards the apex of the ventricle, two from the anterior wall, and the third, which is the shortest from the septum. In many hearts this third column is absent, the *chordæ tendineæ* in that situation arising directly from the septum. The right ventricle has two openings communicating with it, viz.: the auriculo-ventricular at the centre of its base; and that of the pulmonary artery at the distal extremity of the infundibulum, and placed, with relation to the former, anteriorly, superiorly, and to the left side. The auriculo-ventricular orifice is provided with a valvular apparatus, which permits the flow of blood from the auricle into the ventricle, but effectually precludes reflux from the ventricle to the auricle in the normal state. This valve usually consists of three segments, and is hence named *tricuspid*; but, according to Pettigrew,\* in at least thirty per cent. of human hearts it is composed of two segments only, the representative of the third being rudimentary. When three segments are present, one, the largest, is situated anteriorly and somewhat to the left, between the auriculo-ventricular and arterial openings; this is the *septum of Lieutaud*, to which the function of closing the arterial orifice during ventricular diastole was for a long time attributed; another is placed on the right side; and the third and smallest posteriorly and to the left, upon the *septum ventriculorum*.† The structure of these valves, and their connection with the *musculi papillares* and *chordæ tendineæ*, demand a

\* *Transactions of Royal Society of Edinburgh*, vol. xxii. part iii.

† Haller (*Elementa Physiologiæ*, tom. i., p. 291, *et sequent*) assigns to the three segments of the tricuspid valve, the relative positions, "anterior and superior," (septum of Lieutaud) "anterior and inferior," and "posterior," and, with the exception of the last, correctly, in relation to the body.

special and detailed description, owing to their intimate bearing upon the mechanism of occlusion during ventricular systole.

The *auriculo ventricular* opening is bounded by a tendinous ring, composed of white fibrous tissue, and possessing in an eminent degree the property of resistance ; it is strongest anteriorly and in the vicinity of the septum, and gives attachment by its inferior and left margin to the tricuspid valve, and by its superior and right, to the fibres of the right auricle ; very few of the fibres of the corresponding ventricle are attached to this ring. The valve forms an unbroken circle,\* in continuity with it around the opening, and then breaks up into three segments, which occupy the relative positions already indicated. These segments are thick at the base and along the centre, and thin at the free extremity and margins, and each is composed of a reduplication of the endocardium, between the folds of which minute bands of white fibrous tissue are disposed in a definite manner.† According to Pettigrew, each branch of the bifid papillary muscles, which correspond in number with the segments of the valve, gives off the typical number of three chordæ tendineæ, each of which in turn breaks up into three, thus yielding in all nine minute tendons, which are attached to the dorsal or ventricular surface of the adjacent half of the corresponding valve ; the three strongest and most superficial, at equal distances along the middle line from base to apex ; the three next in point of thickness and length, to that portion of the valve intermediate between the mesial line and margin in a similar manner ; and the three shortest and finest, which are likewise in situation the deepest or most remote when viewed from the ventricular aspect of the valve, are implanted into its margin, extending from the attached to the free extremity. A similar arrangement obtains for the tendons arising from the second branch of the papillary muscle,

\* In *Quain's Anatomy*, edited by Professors Sharpey, Thomson, and Cleland (seventh edition, 1867, vol. i.), the learned editors deny the existence of distinct tendinous rings at the auriculo-ventricular orifices ; they assert that the so-called rings consist of bands of loose white fibrous tissue, strengthened anteriorly and towards the septum, by processes derived from the plate of fibro-cartilage, occupying the triangular interval between the aortic and the two auriculo-ventricular openings.

† Vesalius taught that the tricuspid valve consisted of a single layer of membrane.

but which are destined for the adjacent half, not of the same, but of a neighbouring valve. At the opposite extremity of both valves, when two only are present, another papillary muscle with its two crops of tendons, is similarly disposed; but when three valvular segments exist, a third papillary muscle with its tendons, is supplied for the adjacent surfaces and margins of the second and third segments. The tendinous chords arising from a common origin, and destined for the same valve, mutually intersect at various angles, and seem to form an intricate interlacement, owing to their peculiar arrangement in layers and the varying obliquity of their courses; they all expand between the layers of the endocardium, those from opposite sides of the same segment being fused together, so as to form a dense and continuous stratum, constituting its bulk, and giving it strength and consistence. Two or three minor lobes, according to the number of the larger segments, but unprovided with papillary muscles or tendons, are occasionally interposed between the angles of the latter.

From the arrangement thus briefly described, it is manifest that each segment of the auriculo-ventricular valve is supported upon every point of its ventricular surface by two sets of tendons, proceeding from two distinct papillary muscles; that each is; therefore, influenced by two of these muscles; and, further, that each muscle acts upon two valves, and must, of necessity, in contraction, approximate their margins, and in the same proportion, tend to close the passage which they guard. Thus, these muscular columns, whose use has for a long time constituted a problem of great difficulty in physiology, perform a double function, mainly that of sustaining the auriculo-ventricular valve through the chordæ tendineæ, and of preventing its retroversion during ventricular contraction; for, being themselves offsets from, and contracting simultaneously and in an equal ratio with, the muscular walls of the ventricle, they neutralise the relaxing effect upon the valve, which must otherwise result from the gradual approximation of the apex towards the base of the heart in the progress of ventricular systole. They likewise, by their contraction, approximate the free margins of the valves, owing to the connection of *each* of



them with *two* segments, although of themselves they seem incapable of effecting complete closure, notwithstanding the high authority of Reid\* and Bouillaud† to the contrary.

At the first moment of relaxation of the ventricles, the blood, now subjected to the pressure of distension in the auricle, rushes with considerable force into the ventricle, divaricating the segments of the valve, and pressing them against the ventricular walls. During the progress of ventricular diastole, however, the valves are displaced from the walls of the ventricle, and floated athwart the orifice by the accumulating blood, in such a manner as not entirely to occlude it, or prevent the further entrance of blood. The ventricle being now all but fully distended, and the pool of blood in the auricle and ventricle continuous through the partially open auriculo-ventricular orifice, the active contraction of the auricle propels a further volume of blood into the ventricle, completely distending it, and determining its immediate contraction. The pressure to which the blood is now subjected by the muscular walls of the contracting ventricle at all points of its surface, save at its base, where the walls are non-contractile, determines a movement of the mass in this latter direction, or towards the point of least resistance, with a force equivalent to the pressure exercised, which Hales‡ has estimated for the left ventricle at 7·5 feet of a vertical column of blood.

For the right ventricle, according to the calculation of Haughton, the estimate should be somewhat more than one third of the preceding.§ This pressure is borne mainly by the auriculo-ventricular valve, which is forcibly and rapidly closed, whilst the attached chordæ tendineæ, previously relaxed, are put suddenly upon the stretch,|| yielding with the valves a tension-sound, which constitutes the principal element of the first sound of the heart. The attachment of two adjacent segments, at both extremities, to the same papillary muscle, through the chordæ tendineæ, must determine a movement of mutual approximation

\* *Cyclopædia of Anatomy and Physiology* : article "Heart."

† *Traite Clinique des Maladies du Cœur*.

‡ *Statical Essays*, London, 1769, vol. ii.

§ *Principles of Animal Mechanics*, 1873.

|| Haller (*opus citat*, tom. i. liber. iv., cor.) erroneously asserts that during ventricular contraction the chordæ tendineæ are relaxed.

of their free margins under pressure from behind. I cannot by any means admit with Dr. Bell Pettigrew\* that "during the systole, and towards the termination of that act, the valves are, by the contraction of the muscoli papillares, dragged down by the chordæ tendineæ into the ventricular cavities to form two dependent cones;" nor with Professor Halford, that they are projected into the auricles. On the contrary, I believe that throughout ventricular systole the valves present a plane surface towards either chamber, and that, as already stated, the principal function of the papillary muscles is to neutralize, by their contraction, the relaxing effect upon the chordæ tendineæ which must otherwise follow from the approach of the apex towards the base of the heart in the progress of ventricular systole. Neither can I admit, with the first-named distinguished anatomist,† that during ventricular systole the auriculo-ventricular orifice undergoes contraction, irrespectively of the approximation of its valves; nor, that a greater degree of blood pressure is requisite for divaricating the arterial valves than for closing those of the auriculo-ventricular openings.

The orifice of the pulmonary artery is situate at the distal extremity of the infundibulum; it is circular in figure, and surrounded by a tendinous ring of greater strength and thickness than that of the auriculo-ventricular opening,‡ and guarded by a valve, the *sigmoid* or *semilunar*, consisting of three segments, one being placed in front, another posteriorly and to the right, and a third posteriorly and to the left side.§ This orifice has an average circumference in the adult male of three inches and four lines, and in the adult female of three inches three lines; whilst the appended valves, detached and expanded in close juxtaposition, present a united diameter somewhat in excess of that of the orifice which they guard. The tendinous ring which encircles the arterial orifice is composed of three festoons, convex

\* *On the Relations, Structure, and Function of the Valves of the Heart in Vertebrata, Transact. Royal Society of Edinburgh*, vol. xxiii., part iii.

† *Opus citat*, p. 797 and 799.

‡ Luton (*Nouveau Dictionnaire de Médecine et de Chirurgie Pratique*, Paris, 1868) asserts that the auriculo-ventricular zones are stronger than the arterial; the left thicker than the right; and the anterior demi-zone stronger than the posterior.

§ Haller (*opus citat*) defines the relative position of the pulmonary sigmoid valves as anterior, posterior, and superior, respectively.

towards the ventricle, and meeting at acute angles, salient towards the artery. Each of these represents about half a circle, the diameter of which is to that of the artery as 1 to 3.

To the upper or concave surface of these tendinous semi-circles the valves are attached by their convex edges, and immediately above them the artery, which is likewise attached by its middle coat to their concave surface but outer edge, expands into three recesses, corresponding in situation, depth, and figure, to the segments of the valve. These are the *sinuses of Valsalva*. When the valves are partially separated from the wall of the artery, the sinuses, bounded externally by the bulging wall of the vessel, internally by the arterial surface of the valves, and inferiorly or towards the ventricle by the curve of the semi-circular tendons, to the inner and outer edges of which, respectively, the valves and the middle coat of the artery are attached, present each the figure of a boat, and are veritable navicular fossæ.

On a line corresponding to the extremities of the tendinous curves, and the upper boundaries of the sinuses of Valsalva, the wall of the artery is uniformly thick, but that portion of it which enters into the sinuses is considerably attenuated. In the triangular interspaces between the adjacent convex edges of the tendinous festoons at the root of the artery, a dense layer of fibrous tissue, continuous with the festoon on either side, and lined by the endocardium, alone constitutes the wall of the ventricle.

The segments of the valve are thick and uniform along the attached border, but thin and double festooned on the free margin. They are composed, like the tricuspid valve, of a reduplication of the endocardium, with fibrous tissue interposed. The arrangement of this latter element deserves special attention. According to Pettigrew,\* a tendinous thread of extreme tenuity passes along the free border of the valve on each side, to meet in the centre, where a nodular thickening exists, known as the *corpus arantii*. From all points of the convexity of this marginal tendon others of still greater tenuity pass off at acute angles, descending obliquely inwards, the most internal intersecting in the middle

\* *Opus citat*, p. 769 *et sequent*, and plate xxviii.

line of the valve, whilst the external descend towards its attached border, intersecting in their course those placed lower down. Between this marginal filament and the next band of magnitude beneath, the valve over an extent of several lines is thin and pliant, and composed only of the two layers of endocardium, with the offsets from the marginal tendon previously described; this is the *lunula*. Beneath this four or five strong tendinous bands arise on each side from the tendon at the attached border of the valve, and, at equal distances apart, course downwards and inwards with a gentle curve, concentric with those of the margins, and branching as they advance, to intersect those from the opposite side along the middle line; and finally, from the lower margin of the valve, at the bottom of the sinus, several small bands pass upwards and inwards on either side of the mesial line, bracing the others, and giving increased thickness and strength to the attached border of the valve.

During ventricular systole the semilunar valves are divaricated against the walls of the artery by the centrifugal pressure of the escaping column of blood; and according to Haller, the purposes of the sinuses of Valsalva is to furnish recesses in which they may be lodged at this moment, out of the course of the blood. The position of the valves, however, above the tendinous ring, and their extreme tenuity, would seem to render such a provision scarcely necessary. It seems more probable that the use of the sinuses, as urged with much emphasis by Pettigrew, has reference to the direction of closure of the valves.

At the termination of ventricular systole, the elastic recoil of the walls of the artery determines a backward movement of that portion of the column of the blood in proximity to the ventricle; by this movement the valves are closed over the opening with a suddenness and force sufficient to elicit a tension-sound, known as the second sound of the heart. The direction of the blood-pressure, by which the valves are thus closed, is so modified by the pouching of the artery at the sinuses, that if the axes of the columns borne by the three valves respectively were prolonged towards the ventricle, they would meet immediately below them in the axis of the passage. Hence the valves, pressed against one another at the moment of closure,



become mutually sustaining, and the force that would tend to effect their retroversion into the ventricle, is converted into an agency by which that accident is averted.

It has been already shown that the diameter of the conjoined valves exceeds that of the orifice, and that the lunulæ are much thinner and more flexible than the remainder of the valve; if to these two conditions be added the shortness of the marginal tendons, by which the free edge of each valve is slung up between the most elevated points of the corresponding tendinous festoon, and the convergence towards the arterial orifice of the lines of blood-pressure from above, it will be manifest that retroversion cannot occur without rupture of the valve; and further, that rupture cannot take place within the area of the lunulæ, which are disposed *dos à dos*, and out of the lines of blood-pressure. Experience proves the correctness of both these conclusions, displacement of one or more of the segments into the ventricle having never been met with in the absence of disintegration or detachment to some extent, and rupture being confined to the central and attached portions of the valves. The absence of an apparatus of muscles and tendons, destined to prevent retroversion of the semilunar valves during arterial systole, such as that previously discussed in connection with those of the auriculo-ventricular opening, may be explained in part by reference to the mechanical obstruction to the transit of the blood which would result from their presence in the narrow channel of the artery, but chiefly by the fact that such a provision is unnecessary, and would be totally inapplicable to the circumstances under which the semilunar valves are brought into action. The force by which the valves are closed in both instances is, no doubt, blood pressure; but in the one case this is the equivalent of the elastic reaction of the artery in the direction of its axis, whilst in the other it is the representative and guage of a much greater force, the active contraction of the walls of the ventricle, operating not only towards a common centre, but with a definite movement of approximation of the apex towards the valves. The form of the artery favours the application of the provision actually made for the support and protection of its valves; and experience

proves that this provision is fully adequate to its purpose, whilst the greater size and power of the ventricle would render such an arrangement not only unsuitable, but insufficient.

*The left ventricle* occupies the posterior, inferior, and left portion of the heart, extending to the apex, which it entirely forms; about one-third of its superficial extent enters into the anterior surface of the heart, the remaining two-thirds constituting the left border, posterior surface, and part of the inferior surface of the organ. In figure it represents a slightly flattened cone, and in section it is ovoid, the long diameter from before backwards, the septum being concave to the left. Pettigrew\* describes the cavity of the left ventricle as circular at the base, oval half an inch from the base, irregularly triangular half an inch still lower down, more decidedly triangular two inches from the base owing to the projection of the muscoli-papillaries, and finally bayonet-shaped at the apex. The walls are thickest at the junction of the base fourth with the apical three-fourths of its length, and here they are equal to thrice the thickness of those of the right ventricle. The endocardium is more opaque, and the columnæ carneæ are smaller, but more numerous and more closely reticulated than in the right ventricle, especially towards the apex; but on the upper portion of the anterior wall and septum they are entirely absent.

The muscoli papillaries are only two in number, both arising in close proximity to the septum, one from the anterior and the other from the posterior wall of the ventricle. They are much thicker, and the chordæ tendineæ are stronger and less numerous than in the right ventricle. At the base the auriculo-ventricular and arterial orifices are situate, the former posterior and to the left, the latter anterior and to the right side, and in much closer mutual proximity than the corresponding orifices in the right ventricle. The left auriculo-ventricular orifice, smaller than the right,† is ovoid in figure, the long

\* *Philosop. Transact.*, part iii. 1864.

† Dr. Peacock estimates the average circumference of the auriculo-ventricular orifice as follows :

	Males.	Females.
Right	4 inches 6 lines.	4 inches 0 lines.
Left	3    „    7    „	3    „    10    „

diameter directed obliquely from before and from the left side backwards and to the right; it is surrounded by a strong tendon, and guarded by a valve, the *mitral*, thicker and stronger than the tricuspid, and consisting of two segments. Of these the larger, interposed between the two openings, is placed with relation to the auriculo-ventricular orifice anteriorly and to the right, whilst the other and smaller segment is posterior and to the left.\*

The muscoli papillaries of the left ventricle are connected through the chordæ tendineæ with the segments of the mitral valve, in a manner in no respect different from the arrangement already described in the right ventricle. The function of the mitral valve is likewise similar to that of the tricuspid; and the explanation already given as to the mechanism of closure of the latter, may be applied, *mutatis mutandis*, to that now under consideration.

The orifice of the aorta is situate at the superior, anterior, and right portion of the ventricle; it is circular in figure, somewhat smaller than that of the pulmonary artery, posteriorly and to the right of which it lies, but separated from it by the septum of the ventricles.† The sigmoid or semilunar valves, three in number, and placed with respect to the opening, one anteriorly and to the right, another anteriorly and to the left, and a third posteriorly, are much stronger, and the *corpora arantii* more prominent than those in connection with the pulmonary artery. The tendinous zone, similarly constructed, is likewise stronger. It is lodged in the anterior angular recess between the two auriculo-ventricular zones, and the triangular interval bounded by all three, is occupied in the ox by a calcareous mass, the so-called *os cordis*, which in the human heart is represented by fibro-cartilage. The sinuses of Valsalva are deeper and larger‡

\* Haller (*opus citat*) says the mitral orifice is oval horizontally, the valves being respectively "superior" and "inferior."

† Peacock's estimate of the mean circumference of the arterial orifices is as follows:

	Males.	Females.
Right (pulmonary)	3 inches 4 lines.	3 inches 3 lines.
Left (aortic)	3    "    0    "	2    "    10    "

‡ Haller (*opus citat*) states that there are no aortic sinuses in the fœtus or infant.

than those of the pulmonary artery; from the two anterior of these sinuses the coronary arteries take origin, the anterior from the left, and the posterior from the right sinus. Brücke maintains that the orifices of these vessels are within range of the valves, and covered by them when raised; whilst Haller,\* Senac,† Bertin,‡ and Morgagni, and most modern authorities, hold a contrary opinion. I agree with the latter, being convinced, from personal examination in the dissecting room and dead-house, of a great number of hearts free from disease of the aortic valves, that Brücke's opinion is incorrect.

To this subject I shall have occasion to revert when discussing the rhythm of the heart.

The muscular fibres of the heart observe an apparently complex arrangement, being disposed in successive layers, each of which affects a different course from those superficial to and those beneath it. Vesalius§ described the fibres of the ventricles as being, some straight, some oblique, and others transverse; the deep fibres taking a course contrary to that of the superficial. He compares the arrangement to a "plait of rushes."

Lower§ believed that from the tendinous rings surrounding the auriculo-ventricular openings, the fibres of the ventricles, disposed in two planes, a superficial and a deep, expanded over the chambers in a spiral manner; a few at the surface run straight from base to apex. The external fibres embrace the two ventricles; those which cover the right ventricle ascend from left apex to right base, and those which cover the left ventricle ascend from right apex to left base. The fibres, though somewhat spirally disposed, are not in unbroken continuity, the arrangement being that of the figure of 8, the fibres which pass in one direction form the external surface, whilst coursing in the opposite direction they line the internal surface. The most superficial fibres only pass to the apex, those more deeply seated turning inwards sooner.

Lancisi§ held that a tendinous ring surrounded each of the four openings of the heart, and that from those of the auriculo-ventricular orifices the fibres arose and passed spirally round

\* *Opus citat*, liber. iv. cor. p. 367.

† *Traite des Maladies du Cœur*.

‡ *Traite de la Structure du Cœur*, p. 365-6.

§ Cited by Senac (*opus citat*).



the ventricles, being interlaced like wicker-work in their course. The fibres of the auricles start from the roots of the *venæ cavæ* and pulmonary veins, and passing over the auricles in two layers, viz., external and internal, end in being attached to the tendons of the auriculo-ventricular openings.

Senac\* describes three layers of fibres in the ventricles. The most superficial arise from the veins and auricles, and pass spirally from base to apex. Several, however, dip into the substance of the heart, and seem to serve as "binding" fibres for others. The principal fibres turn in at the apex and pass upwards, forming the internal surface of the ventricles. The descending fibres incline at different angles to the axis of the ventricles, some at acute, and others at nearly right angles, the latter not reaching the apex, and all return to be attached to the tendinous rings of the auriculo-ventricular openings. Between the descending and the ascending fibres is a third layer similarly disposed, but not traceable to the base. The arrangement is the same for the two ventricles, and their fibres conjointly form the septum.

Winslow maintained that the heart was composed of two muscular sacs enclosed in a third, which was equally muscular. Haller† describes all the fibres of the ventricles as seeming to him oblique, approaching rather the horizontal than the vertical line. Arising in the situation of the right auricle, they are bent round the anterior or convex surface of the heart, to the apex, and thence back to the right auricle along the inferior or plane surface of the heart. Near the base many fibres run inwards towards the cavity to form the *carneæ columnæ*, and hence the walls are thinner at the apex than at the base. The external and internal strata are more oblique, the intermediate fibres more transverse. In the septum cordis the fibres of both ventricles are interlaced, but nearly parallel. The fibres of the *musculi papillaries* are straight, and run from apex towards venous (auriculo-ventricular) openings.

He described the superficial fibres of the left auricle as being transverse, and the deep as oblique, some ascending, some descending, and others surrounding the orifices of the pulmonary veins.

\* *Opus citat.*

† *Opus citat*, liber. iv., cor.

Pettigrew\* has carefully studied and minutely described the arrangement of the muscular fibres of the heart. According to him the ventricles are composed of seven superposed layers of fibres, *i. e.*, three superficial layers, a "middle layer," and three deep layers. The three superficial layers run from base to apex, and from right to left downwards, with progressively increasing obliquity, the first nearly vertical, and the third nearly horizontal, whilst the fourth or middle layer is all but horizontal. These fibres are connected at the base chiefly with the auriculo-ventricular tendinous rings, but partly also with those of the arterial orifices; a few likewise with the columnæ carneæ. They do not, however, arise from those rings, being connected with them only by areolar tissue. Several are inflected from the anterior surface, in the line of the anterior coronary groove, to form the ventricular septum, two thirds of which are formed of the inflected fibres of the left ventricle, and one-third by those of the right ventricle. In the septum the inflected fibres course obliquely upwards. The fibres of the deep layers run from apex to base, and from right to left *upwards*, likewise with progressively increasing obliquity, those of the first (fifth from the surface) being nearly horizontal, and those of the third (seventh from the surface) nearly vertical. The deep fibres are continuous with those of the superficial layers at the apex and base, so as to form a series of concentric ellipses, the seventh with the first, the sixth with the second, and the fifth with the third, respectively. The layers are curtailed in vertical dimensions in progressive ratio from the first to the seventh, so that the extreme apex is composed of only a single layer, viz. the first, the anterior and posterior portion of which are so inflected and mutually involved that the anterior fibres, after curving round, enter the apex at the posterior surface, and become continuous with the posterior columnæ carneæ and papillary muscle; whilst the posterior fibres, after being inflected to the front, enter the apex anteriorly, and become continuous with the anterior carneæ columnæ and papillary muscle. Thus is formed the "vortex,"

\* *On the Arrangement of the Muscular Fibres in the ventricles of the Vertebrate Heart, with Physiological Remarks*, by James Bell Pettigrew, M.D., *Philosop. Trans.*, part iii., 1867.

or "whorl," at the apex of the left ventricle. The aggregate of the columnæ carneæ and musculi papillares constitute the seventh layer, which courses spirally from right to left, and from apex to base.

The right ventricle is moulded on the left. The septum ventriculorum is formed by an inflection of the anterior wall in the line of the anterior interventricular sulcus, and none of the layers cross this groove except a narrow band derived from the superficial layers near the base, whilst posteriorly all the layers cross the groove. Hence, perhaps, the deficiency of the basal portion of the septum ventriculorum in the early stage of embryonic development. The inflected or septal fibres derived from the right ventricle belong exclusively to the three superficial layers; all the more deeply seated fibres being interrupted at the septum. Hence the right side of the septum is much thinner than the left.

The layers entering into the right ventricle are less thick than those of the left, a fact which explains the comparative thinness of the walls of that chamber. The walls of both ventricles are thickest at the centre, owing to the relative vertical dimensions of the successive layers previously described.

The sinuses of the auricles are composed of a superficial layer common to both auricles, and passing horizontally from one to the other, but most distinctly so on the anterior surface. A great portion of this layer is inflected into the interauricular septum posteriorly; and two deep layers, one of which consists of looped fibres connected by both their extremities with the auriculo-ventricular rings, and the other of annular fibres, situate at the venæ cavæ, the coronary sinus, and the pulmonary veins, and constituting a sort of sphincter for these vessels. Upon the venæ cavæ and the pulmonary veins these fibres extend from the auricles nearly half an inch. The walls of the appendices consist of a superficial layer of annular, and a deep layer of vertical fibres, the musculi pectinati already described.

Luton \* describes the fibres of the ventricles as common and proper. The former are the "unitive" fibres of Gerdy, and are disposed on the anterior and posterior surfaces. The anterior

\* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratique*; Paris, 1868.

fibres pass down from right to left, and from base to apex, and are disposed in three ways, being all, however, rolled in at the apex like a whorl, leaving a minute aperture between, through which the ventricle may be entered without dividing any of its fibres. One set, the "simple looped fibres" (Bourguery), pass from the anterior to the posterior wall, and return to the base along the internal surface of the posterior wall, to be inserted into the posterior segments of the aortic and mitral zones. Another set, the "figure of 8" fibres (Bourguery), pass from behind forwards at the apex, course along the inner surface of the anterior wall to the base, where they are likewise attached to the anterior segments of the aortic and mitral zones. The third set passes in to form the columnæ carneæ, and are inserted through the chordæ tendineæ into the segments of the mitral valve.

The posterior "unitive" fibres pass from the posterior segments of the auriculo-ventricular zones, obliquely downwards and to the right edge of the heart, where they enter the right ventricle in succession, along the entire right border, the longest being at the apex, and are thence reflected upwards on the inner surface of the anterior wall, forming "simple loops," to be inserted into the right auriculo-ventricular zone, and that of the pulmonary artery, and also forming columnæ carneæ to be inserted through the chordæ tendineæ into the tricuspid valve.

The proper fibres of the ventricles are disposed in loops attached to the arterial and auriculo-ventricular zones, and form cylinders, open at the apex (but here narrow), and at the auriculo-ventricular orifice, and interposed between the descending and ascending layers of the "unitive" fibres. The septum is formed of fibres derived from both ventricles, but chiefly the left.

The auricular walls are likewise composed of two layers of muscular fibres, viz., a common and a proper.

The muscle of the heart, according to Kölliker,\* differs from voluntary muscle in the following particulars, viz., the fibres are only one third the size of those of voluntary muscle; the longitudinal striæ are occasionally better marked than the transverse; they are more easily broken up into filaments and frag-

\* *Traité d'Histologie Humaine.*



ments. The sarcolemma is difficult of demonstration, save by reagents. The primitive fasciculi are often infiltrated with fatty granulations disposed in lines, a condition which exists in an exaggerated form in fatty degeneration. The fibres are not, as in voluntary muscle, disposed in fasciculi of different orders, but are closely packed, with very little connective tissue, and are connected with one another by branched fibres, which justifies the conclusion that they contract simultaneously. I have found the average diameter of the fibres to be 1-1300 of an inch ; but the linear arrangement of highly refractile granules within the fibres I have not met with, save in the early stage of fatty degeneration. This "granular" condition of the fibres is regarded by Ormerod \* as precursory to fatty transformation.

The arteries of the heart, named *coronary* from their peculiar disposition in the form of circlets or chaplets, are usually two in number, the *right* or *posterior*, and the *left* or *anterior*. They arise from the aortic sinuses of Valsalva, immediately above the level of the free edges of the valves when raised against the walls of the vessel. The former from the anterior and right, and the latter from the anterior and left sinus.

Occasionally both vessels arise by a common trunk, which subdivides into two branches, representing the usual arteries. Still more rarely there are three, or even four parent trunks, the supernumerary vessels arising as duplicates from the anterior sinuses, but smaller in size than the representatives of the usual vessels.

The *right coronary artery* descends between the root of the aorta and the right auricular appendix ; it then enters the right auriculo-ventricular groove, passes horizontally to the right, and divides into two branches. Of these the smaller descends obliquely to the left, along the right border of the heart to the right apex, whilst the larger or horizontal branch continues in the auriculo-ventricular groove, to the posterior interventricular sulcus, where it undergoes further division into two branches, viz., a vertical or larger, which descends in that groove to the apex, and a horizontal, which terminates by anastomosing with the horizontal branch of the left coronary artery. In its course the

\* *St. Bartholomew's Hospital Report*, vol. iv., 1865.

trunk of the right coronary artery supplies small branches to the right auricular appendix, pulmonary artery, right auricle and ventricle, whilst its branches are expended in supplying the right auricle and ventricle, and the posterior part of the septum ventriculorum.

*The left coronary artery* descends obliquely to the left, between the pulmonary artery and appendix of the left auricle, to the anterior interventricular groove, where it undergoes division into two branches, the larger of which descends in that groove to the right of the apex, where it inosculates with the vertical branch of the right coronary,\* whilst the smaller takes a horizontal course in the left auriculo-ventricular sulcus, to anastomose posteriorly with the corresponding branch of the right artery. This branch supplies by its trunk some branches to the aorta, pulmonary artery, and left auricular appendix, and by its two branches of division, the left auricle and ventricle and the anterior portion of the septum.

*The coronary veins* are divisible into the *great cardiac* or *coronary vein*, and the small coronary veins. The great coronary vein commences at the apex of the heart, whence it ascends by the anterior interventricular furrow, receiving in its course superficial and deep branches from both ventricles, and from the septum. At the base of the ventricles it becomes horizontal, passing in the left auriculo-ventricular groove to the posterior surface of the heart, and opening into the right auricle between the orifice of the inferior vena cava and that leading into the right ventricle. This vessel, in the terminal portion of its course, is remarkable for its large size, and named the *coronary sinus*; it receives tributary branches from the left auricle and ventricle; amongst the latter is a large branch which ascends along the left border of the heart to join the principal trunk at a right angle, and a still larger vein, the posterior interventricular, which ascends from the apex by the posterior interventricular groove, and opens into the coronary sinus at its termination.

\* Hyrtl (*Nat. Histor. Review*, 1861, p. 321) as the result of a series of experiments with injection, declares that the communication between the right and left coronary arteries, if it exist at all, is not such as to admit of the passage of ordinary injection material from one to the other.

The coronary veins are unprovided with valves, save at their openings into the sinus. The communication of the sinus with the right auricle is guarded by the valve of *Thebesius* already described. Cruveilhier\* denies the alleged competency of this valve to prevent reflux, because, in his experience, injection thrown into the auricle by the superior vena cava has always found entrance into the coronary sinus. The contraction of the auricle, however, at the only period when the contained blood is subjected to pressure equivalent to that of an injecting syringe, is probably sufficient to insure the closure of the orifice and prevent reflux. The small coronary veins are those which have been previously described as conveying blood, chiefly from the right side of the heart, and opening by minute orifices into the right auricle.†

The *nerves of the heart* are derived immediately from the cardiac plexuses, two in number, viz., the superficial and the deep. These plexuses are formed by the union of the *cardiac* branches derived from the three cervical ganglia of the sympathetic, and those from the pneumogastric and the recurrent laryngeal nerves.

The cardiac nerves from the sympathetic are three in number, the superior, middle, and inferior. The *superior* or *superficial* cardiac nerve arises by two branches from the superior cervical ganglion, and by an equal number from the sympathetic cord below it; it descends the neck, resting on the longus colli muscle behind the carotid sheath, and inside the middle cervical ganglion; at this point it gives off a large branch which undergoes division into three. One of these crosses over the inferior thyroid artery, and descends to the thyroid body, whilst the two remaining branches embrace the common carotid artery, and descend on its anterior surface to join the first cardiac branch of the pneumogastric. The nerve then passes across the inferior thyroid artery and recurrent laryngeal nerve, and on the right side enters the chest either before or behind the subclavian artery, and is conducted by the arteria innominata to the arch of the aorta, behind which it passes to join the posterior cardiac

\* *Descriptive Anatomy*, vol. ii., p. 766.

† Senac denies that any of these veins open into the left side of the heart.

plexus, giving off a few branches which twine around to the anterior surface of that vessel.

The nerve of the left side enters the chest, resting on the left common carotid artery, and crosses the arch of the aorta anteriorly or posteriorly (usually the former) to enter the anterior cardiac plexus. These nerves, in their course, communicate with the external laryngeal, the pneumogastric, the middle cervical ganglion, and the recurrent laryngeal.

The *middle* cardiac nerve, also named *deep* and *great* cardiac nerve, arises from the lower and inner angle of the middle cervical ganglion; it then descends behind the carotid sheath, and enters the chest on the right side by passing either in front of or behind the subclavian artery\*; and on the left, between the carotid and subclavian arteries. In its course down the neck it communicates with the superficial cardiac, and the recurrent laryngeal nerve, and after entering the chest follows the course of the trachea, by which it is conducted to the deep cardiac plexus.† In the thorax, the deep cardiac communicates with the recurrent laryngeal and the superficial cardiac nerves, the connection with the latter being established, on the right side, on the posterior and external surface of the innominate artery.

The *inferior* cardiac nerve arises from the lower cervical, frequently also in part from the first thoracic ganglion; on the right side it descends behind the subclavian artery; and on the left, in company or in union with the middle cardiac nerve, to enter the deep cardiac plexus.

On both sides this nerve communicates with the middle cardiac and the recurrent laryngeal; and on the right, this is observed to take place behind the subclavian artery.

The cardiac branches of the pneumogastric are derived in part directly from that nerve, and in part from the recurrent laryngeal. The former are, in the neck, *superior* and *inferior*. The *superior* cardiac branch or branches of the pneumogastric arise from that nerve about the level of the thyroid cartilage, and after a short course downwards, are lost by uniting with the

\* Scarpa (*opus citat.*) describes it as invariably passing in front of the subclavian artery.

† According to Scarpa (*opus citat.*) it is lost in the cardiac ganglion of Wrisberg.



sympathetic cardiac nerves.\* The *inferior* cardiac is a nerve of considerable size, arising from the pneumogastric at the lower part of the neck; on the right side it enters the chest on the outer side of the innominate artery, and joins one of the cardiac branches of the sympathetic, which it accompanies into the deep cardiac plexus. The inferior cardiac nerve of the left side passes in front of the aorta to enter the superficial cardiac plexus. Finally, within the chest, the right pneumogastric gives off, whilst lying on the side of the trachea, a few small branches which descend to the deep cardiac plexus; but on the left side these branches come from the recurrent laryngeal; which nerve also gives off on the right side several cardiac filaments from the convexity of its curve.

The *superficial cardiac plexus* lies within the curve of the arch of the aorta, and in front of the right branch of the pulmonary artery. It is formed from the left superior or superficial cardiac nerve, which ends in it wholly or in greater part; and the inferior cardiac branch of the left pneumogastric, and occasionally also that of the right side. At the point of union of the two nerves which contribute to form this plexus, a small gangli-form enlargement, the *cardiac ganglion of Wrisberg*, is occasionally found. This plexus ends in the anterior coronary plexus; but from it pass numerous lateral branches in front of the left pulmonary artery, to the left anterior pulmonary plexus.

The *deep cardiac plexus* lies between the transverse portion of the arch of the aorta, and the bifurcation of the trachea, above the point of division of the pulmonary artery. In it terminate the cardiac branches of the cervical sympathetic, except the left superior; and those of the pneumogastrics, except the left inferior; besides those derived from the recurrent laryngeal nerves of both sides. From the right side of this plexus a great number of branches descend in front of the right pulmonary artery, to join the branches from the superficial plexus in the

\* Scarpa (*opus citat.*) describes two superficial cardiac nerves as arising from the pneumogastric on each side; the superior a short distance above the subclavian artery on the right side, and passing down in front of that vessel at its origin; the inferior likewise above the subclavian artery on the right, and entering the chest in front of it.

formation of the anterior coronary plexus; from it, likewise, some branches pass behind the pulmonary artery, to supply the right auricle, and a few to join the posterior coronary plexus; and from its left side a few filaments pass along the *ductus arteriosus* into the superficial plexus, whilst the greater number of branches enter the posterior coronary plexus. From both sides of the deep plexus several branches are given off to the anterior pulmonary plexuses.

The *coronary plexuses* correspond in number and position with the coronary arteries, the course of which they follow on the surface of the heart.\* The *anterior* coronary plexus is derived primarily from the superficial cardiac; it is placed between the pulmonary artery and aorta, below which point it is reinforced from the deep cardiac, and sends off numerous branches which accompany the anterior coronary artery in its course.

The *posterior* coronary plexus is formed chiefly from the left, but receives a few branches from the right extremity of the deep cardiac plexus. It accompanies the posterior coronary artery to the posterior surface of the heart.

Nerve filaments from the coronary plexuses accompany the branches of the coronary arteries, not only on the surface, but likewise into the substance of the heart. They are distributed in the muscular tissue of the organ, and form a fine net-work beneath its lining membrane, easily seen in the heart of the lamb.

These nerve-fibres freely intercommunicate in the substance of the heart, and at their points of junction minute gangliform enlargements, first described by Remak,† may be observed. These ganglia are dispersed everywhere through the muscular tissue of the heart, but may be found in greatest number in the interventricular and the auriculo-ventricular septa. Three of these demand special notice, viz., the ganglion of Remak *par excellence*, near the opening of the inferior vena cava; that of Bidder near the mitral orifice; and the ganglion of Ludwig in the wall of the right auricle.

\* Scarpa (*Tubulæ Neurologicæ*, Ticini, 1794, *passim*) in his description of the coronary arteries and plexuses, transposes both. Thus, he describes the right coronary artery and plexus as *anterior*, and the left as *posterior*.

† Müller's *Archiv*. 1844.

The sub-pericardiac ganglia (so called) of Lee are generally regarded as only thickenings of the neurilemma. To the ganglia in the septa between the auricles and ventricles, in the immediate vicinity of the auriculo-ventricular openings, the experiments of Paget have directed special attention, as intimately associated with the rhythmic action of the heart.

The *lymphatics* of the heart are deep and superficial. The former permeate the substance of the heart and terminate in the latter, which follow the course of the blood vessels from the apex towards the base, and there form by their union two large trunks, one for the right, and another for the left side. The right lymphatic trunk ascends, resting upon the aorta, and crosses the transverse portion of the arch between the innominate and the left carotid arteries, to the trachea, and thence to the right lymphatic duct, which it enters.

The left trunk courses along the trunk of the pulmonary artery to its bifurcation. It then passes behind the arch of the aorta to the trachea, which conducts it into the neck, where it opens into the thoracic duct near its termination. These two trunks in their course along the aorta and pulmonary artery, form, each, by their frequent division and reunion, a chain rather than a single vessel.

The *dimensions, weight, capacity, and thickness* of the heart, and its various parts, are subject to such considerable variety, even within the limits of health, that it is difficult, and in the opinion of many authors impossible, to fix a standard of comparison. The following estimates are given as the *means* of a great number of experiments by the subjoined authors.

Measures of size are given in inches and decimals of an inch ; measures of capacity in ounces and decimals of an ounce.

Bizot's measurements, with the exception of the depth of the sigmoid valves, are given as rendered into British measure in Dr. Stokes's work on *Diseases of the Heart and Aorta*.

	Bouillaud.	Bizot.	Gendrin.	Cruveilhier.	Lobstein.	Marshall.	Quain and Sharpey.	Reid.	Peacock.	Hayden.
Length, ...	3·62 in.	{ 3·82 in. male 3·64 in. female }	{ 3·93 in. 8·5 oz. }	—	5·5 in.	{ 5 in. male 10 to 12 oz. female }	{ 5 in. — }	—	—	4·25 in.
Weight, ...	8·37 oz.	—	—	7 to 8 oz.	9 to 10 oz.	{ 8 to 10 oz. }	—	{ male 11 oz. male 9·75 oz. female 9 oz. female 9 oz. }	—	9 to 10 oz.
Thickness:		At base.								
Right ventricle ...	0·21 in.	Male. Female. 0·16 in. 0·15 in.	0·20 to 0·31 in.	—	0·18 in.	—	—	—	—	0·25 in.
Left ventricle, ...	0·58 in.	0·43 in. 0·36 in.	0·39 to 0·47 in.	—	0·58 in.	—	—	—	—	0·50 in.
Right auricle, ...	0·08 in.	—	—	—	0·04 in.	—	—	—	—	0·08 in.
Left auricle, ...	0·12 in.	—	—	—	0·08 in. at base	—	—	—	—	0·12 in.
Capacity:		Length.								
Right ventricle, {	a pullet's egg	Male. Female. 3·33 in. 2·97 in.	—	right some- what less than left	—	—	2 oz.	—	—	3 oz.
Left ventricle	a little less	Breadth. 7·40 in. 6·80 in.	—	—	—	—	2 oz.	—	—	3 oz.
		Length. 2·61 in. 2·83 in.	—	—	—	—	—	—	—	—
		Breadth. 4·72 in. 4·10 in.	—	right a little larger than left	—	—	—	—	—	with ends of veins super- added, equal to ventricles.
Auricles, {	equal to ven- tricles, right $\frac{1}{2}$ larger than left	—	—	—	—	—	—	—	—	—
Transverse diameter	{ 3·63 in. below base }	—	—	—	3 in. at base	3·5 in.	3·5 in.	—	—	3·5 in.
Circum. at base of ventricles, {	8·75 in.	—	—	—	—	—	—	—	—	—
Circum. of orifices:										
Right auriculo- ventricular, {	3·83 in.	male 4·81 in. female 4·18 in.	—	—	—	—	—	—	{ male 4·50 in. female 4·00 in. }	{ 5·49 in. 3·92 in. }
Left auriculo- ventricular, {	3·54 in.	male 4·29 in. female 3·61 in.	—	—	—	—	—	—	{ male 3·58 in. female 3·83 in. }	{ 2·74 in. 2·35 in. }
Right arterial, ...	2·66 in.	male 2·79 in. female 2·60 in.	—	—	—	—	—	—	{ male 3·25 in. female 3·17 in. }	{ 2·35 in. 0·46 in. pul. a. }
Left arterial, ...	2·42 in.	male 2·74 in. female 2·49 in.	—	—	—	—	—	—	{ male 2·83 in. — }	{ 0·41 in. aortic 0·41 in. }
Depth of sigmoid valve	—	0·55 in. At middle.	—	—	—	—	—	—	—	—
Thickness of inter- {	0·01 in.	male 0·43 in.	—	—	—	—	—	—	—	—



Doctor Herbert Davies,\* in a communication to the Royal Society, lays down a law of fixed relative proportion between the areas of the orifices on the two sides of the heart, which may be briefly stated as follows, viz.: As an indispensable condition to the maintenance of the circulation, and the avoidance of stasis, equal volumes of blood must pass through the corresponding orifices on the two sides of the heart in the same time, or during the systole of the auricles and of the ventricles respectively; but as the systemic is more extensive, and liable to greater and more numerous obstacles than the pulmonic circulation, it must be effected with greater velocity and force than the latter, in order to be accomplished within the same time, and with equal certainty, and the left ventricle must exceed the right in thickness in an equal proportion; but in order that the same volumes of blood shall pass through corresponding orifices within the same period of time, but with different velocity and force, the areas of the orifices must differ in a degree correlative with that of the velocity and force of the circulation through them, but *inversely*; and furthermore, the relative proportion of the area of the corresponding orifices on the two sides of the heart is the same; thus, the area of the tricuspid : area of the mitral orifice :: area of pulmonic : area of aortic orifice. The velocity of the streams through the respective orifices is inversely as the areas of those orifices. Through the tricuspid orifice the velocity of the current is 5 inches per second, through the mitral 7 inches; pulmonic, 7·3 inches, and aortic 23·1 inches in the second.

This is a most important communication, as placing upon a proper and scientific basis the relative size of the corresponding orifices, and thickness of the walls of the corresponding chambers on the two sides of the heart.

The proportion which the weight of the heart bears to that of the body has been variously estimated; by Tiedemann it is given as 1 : 160; by Clendinning as 1 : 158 (male), 1 : 149 (female); and by Reid as 1 : 169 (male), 1 : 176 (female).

Bouillaud† states that the walls of the heart increase in

\* *Transactions of Royal Society*, 1870, and Abstract in *Med. Press and Circular*, 27th April, 1870.

† *Traite Clinique des Maladies du Cœur*, tome premier, 1835.

thickness in a progressive ratio from 16 to 40 years, being thicker in persons of strong build and tall stature than in those of the opposite conformation.

According to Reid\* the heart increases in weight during the entire period between 16 and 70 years, being in the male 8oz. 10dr., and in the female 6 oz. 13dr. at the first-mentioned age, and 12oz. 6dr. and 9oz. 5dr. respectively, at the age of 70 years. Peacock's† estimates for these ages respectively, in the two sexes, are, male 8oz. 2 $\frac{2}{3}$ dr., female 8oz. 1 $\frac{2}{3}$ dr.; and male 10oz. 13 $\frac{1}{3}$ dr., female 7oz.

Bouillaud estimates the weight of an *atrophied* heart as in the mean 175 grammes below the standard, and an *hypertrophied* heart as having a mean excess in weight of 211 to 298 grammes. The most hypertrophied heart, he adds, is more than five times the weight of the most atrophied, and nearly thrice that of the normal heart. The heaviest heart which he has met with weighed 21 $\frac{1}{2}$ oz. Lobstein has seen one which weighed 32oz. after removal of clots. I have in my possession two hearts, to be referred to in the sequel, each of which weighs over 32oz.; one of these was removed from the body of a boy under eighteen years of age. Dr. Stokes‡ has exhibited to the Pathological Society a heart, with adherent pericardium, which weighed 4lbs. 2oz.

The following graphic account of the *development* of the heart and great vessels is taken from *Quain's Anatomy* :§

“The heart first appears as an elongated sac or dilated tube lying at the forepart of the embryo, having two veins connected with it behind, and a large arterial trunk proceeding from it in front. This tube exhibits rhythmic contractions of its walls from a very early period. Its form is at first symmetrical, but soon it becomes curved or bent upon itself like a horse-shoe, and projects on the ventral aspect of the body towards the right side.

\* *Lond. and Edinb. Monthly Jour. of Med. Science*, April, 1843.

† *Ib.*, 1846, and subsequently (1854) printed in separate form.

‡ *Pathol. Society (Dublin) Reports*, 1868-9.

§ *Elements of Anatomy*, edited by Drs. Sharpey, Allen Thompson, and Cleland, 1867, vol. i. p. 323.

“As this bending increases, the venous end approaches the arterial, and at the same time the tube, which progressively increases in size and in the thickness of its walls, becomes divided by two slight constrictions into three compartments, opening successively into each other. The first, next to the veins, is the *auricular* portion, the middle one is the *ventricular*, and the last, which is the primitive arterial trunk, is named the *bulbus arteriosus*.

“The auricular portion becomes placed behind the ventricular compartment, and relatively to that cavity, considerably enlarged. Moreover, two little pouches appear upon it, one at each side, which form the future auricular appendages. The walls of the ventricular portion are already thicker than the rest.

“The next series of changes consists in the gradual subdivision of the single auricle, ventricle, and arterial bulb, each into two compartments, to form the right and left auricles, the right and left ventricles, and the pulmonary artery and aorta; and these changes are accompanied by an alteration in the position of the parts with relation to the body, the ventricular portion now lying transversely, so as to bring that portion which is afterwards to form the apex, towards the left side.

“This subdivision commences first in the single *ventricular* portion of the heart. A small notch appears externally to the right of the apex, which goes on increasing in depth for some weeks, and then is again gradually obliterated. In the meantime, about the fourth or fifth week, a septum begins to rise up internally from the right side of the heart, at a little distance from the apex, and from the anterior wall of the cavity, and proceeds in the direction of the base, towards the arterial bulb, and about the eight week is complete. Traces of the subdivision of the auricular portion commence early in the form of a slight constriction on the outer surface, which marks off the future auricles, the left being at first the smaller of the two but the auricular septum is not begun until after that of the ventricles is completed. About the ninth week it appears, growing from above and behind downwards and forwards, and at length comes to meet and coalesce below with the rising edge

of the interventricular septum. The interauricular septum, however, remains incomplete during intra-uterine life, and leaves an opening in the middle which forms the *foramen ovale*. The farther steps in the separation of the auricles are connected with the changes which take place at the entrances of the great veins. There are now three large vessels terminating in the auricular extremity of the heart. Of these, two correspond with the superior and the inferior vena cava, and the third is the great coronary vein. At first, after the interauricular septum is partly formed above, the inferior cava opens directly into the left auricle, which is the smaller of the two, but about the twelfth week a septum, *the valve of the foramen ovale*, which afterwards forms the floor of the fossa ovalis, rises up on the left side of the entrance of the vein, which thus comes to open into the right auricle; whilst at the same time the separation of the two auricles is also rendered more complete by the gradual advance of the valve over the foramen ovale, leaving, however, the passage open until after birth.

“Another valvular fold is developed at an early period on the right and anterior border of the orifice of the inferior cava, between it and the auriculo-ventricular orifice; this is the Eustachian valve. It appears to continue the opening of the inferior cava towards the upper margin of the foramen ovale, and directs the blood of the vein through that passage.

“The left auricle has at first no connection with the pulmonary veins. The manner in which the connection is afterwards established has not yet been ascertained.

“Originally the heart is composed of a mass of nucleated cells, similar in character to those which primarily constitute the other organs of the body. Muscular tissue is subsequently formed from these cells; but the rhythmic contractions commence and proceed for some time, whilst the heart is yet composed of cells, and before the muscular fibres have been developed.

“*The Great Vessels*: At first the bulbus arteriosus is divided into two arches, which pass upwards and outwards, one on each side, then turn downwards, and form a right and left root of the aorta, which are at first separate, but afterwards unite behind the



heart and in front of the vertebral column, to form the single stem of the descending aorta. The distance soon elongates between those arches and the arterial bulb, and four other pairs of arches appear in series from above downwards, passing outwards from the vessel which ascends to the first arch, and opening into that which descends from it. Thus there are on each side *five arches*, an *internal* or *anterior* trunk uniting the origins of the arches, and an *external* or *posterior* trunk uniting their terminations, and continued into one of the *roots of the aorta*. These vascular arches are placed each in one of the branchial processes of the dorsal plates, but it is to be noted that the whole five arches do not co-exist; for the highest disappear before the last are developed. This arrangement of blood-vessels, together with the originally single condition of the heart, corresponds to a certain extent with the permanent condition of the heart and branchial arteries in fishes, with difference as regards the vascular arches in the human foetus, and that of mammals, birds, and scaly reptiles, that they never present any further branchial subdivision.

“As the interventricular septum is approaching the base of the heart, that is, about the seventh or eighth week, the arterial bulb becomes also divided by an internal partition, meeting from opposite sides, into two vessels, which are slightly twisted on each other, and are so adjusted as to become connected, the anterior with the right, and the posterior with the left ventricle; these vessels afterwards constitute the commencement of the pulmonary artery and of the aorta. A furrow subsequently, beginning on the outside, completes the separation into two vessels.

“Whilst the arterial bulb is thus converted into the commencement of the pulmonary artery and aorta, the five vascular arches arising from it undergo a metamorphosis, by which the permanent aorta, with the brachio-cephalic vessels and the pulmonary arteries, are formed. The general results of this change have been observed by several embryologists, but it has not yet been made out with certainty in all its details.

“It is generally admitted, however, that the fourth arch on the left side (counting from above), which receives blood from the

aortic division of the bulb, is persistent, and continuing to enlarge, eventually becomes the arch of the aorta. The fourth arch on the right side, as well as the first, second, and third arches on both sides, are obliterated to a greater or less extent, while certain portions of them, remaining pervious and connected with the aortic arch, appear to form the commencement of the great vessels rising from it.

“Both the arches of the fifth pair were held by Baer to be connected with the pulmonary division of the bulb, and to send ramifications into the lungs, so as to form the right and left branches of the pulmonary artery respectively; the further or distal portion of the right arch being obliterated, while the corresponding part of the left side continued open, as the *ductus arteriosus*, until birth. According to this view, the third arch on each side is persistent as the subclavian artery, and the external trunk above this remains as the vertebral artery, and the internal as the carotid; while the internal trunk between the third and fourth arches of the right side becomes the innominate artery. In so far as it applies to birds and some reptiles this view may be correct. But a different view of the metamorphosis, as it occurs in mammalia and man, has more recently been presented by Rathke, which has been adopted by Kölliker and others, and probably is more consistent with truth. According to Rathke, in man and mammalia one arch only, viz., the left fifth, is concerned in the formation of the pulmonary arteries; and the fifth arch of the right side is entirely obliterated. From the fifth left arch a branch is given off, which, together with the proximal part of the arch, forms the pulmonary artery, and which divides into the primary branches for the right and left lung, the distal part of the arch being converted, as according to Baer's theory, into the ductus arteriosus. The fourth arch of the right side, according to Rathke, forms the commencement of the right subclavian artery; a branch is given off opposite the external extremity of the fourth arch on both sides, which forms on the right side the remainder of the right subclavian, and on the left the whole of that artery; the vertebral arteries are derived from the subclavians external to the system of arterial arches; the internal trunks in their extent between the third

and fourth arches remain as the common carotids, and in the remainder of their extent form the external carotids, while the third arches and the external trunks above them are converted into the internal carotid arteries."

The *heart of the fœtus* occupies a vertical position in the body till about the fourth month, when it commences to undergo a partial revolution on its antero-posterior axis, by which the apex is carried to the left of the mesial line, and the long axis of the organ becomes oblique in relation to it. Again I quote from *Quain's Anatomy*.\* "For a long period the auricular portion is larger than the ventricular, and the right auricle is more capacious than the left; but towards birth these peculiarities disappear, and the ventricular portion becomes the larger part of the heart. As to the ventricles themselves, the right is at first the smaller; afterwards it becomes the larger of the two, and at birth their size is about equal. In the right ventricle the infundibulum is at first less marked than afterwards.

"For a time the walls of the ventricles are comparatively speaking very thick, and the thickness of both is nearly the same."

The comparatively greater thickness of the walls of the right ventricle in the foetal heart, than in that of the adult, is due to the circumstance that in foetal life the right ventricle has imposed upon it in great part, the function of propelling the blood through the vessels of the lower half of the body, and likewise through the extensive and tortuous circuit of the placental system; whereas, during extra-uterine existence, the pulmonary circulation alone is maintained by this chamber. The salient distinctive features of the foetal heart are, the direct communication between the right and left auricles through the foramen ovale, the patency of the ductus arteriosus, and the large size of the Eustachian valve.

The *foramen ovale* occupies the lower and posterior part of the interauricular septum; it attains its greatest magnitude about the sixth month of foetal life, is bounded by a smooth and rounded muscular rim, from the lower and posterior portion of which a valvular fold, the *operculum*, of a crescentic figure, and consist-

\* Vol. i. p. 327.

ing of a duplicature of the endocardium with some intervening fine elastic tissue, ascends. The operculum adheres to the lateral boundaries of the foramen, but superiorly it is unattached, and here it presents a concave margin, which, although overlapping the superior boundary of the foramen, being placed on the left side of the septum, admits the passage of a current from right to left, but resists one in the opposite direction. Senac\* correctly holds that the foramen ovale is kept open in the fœtus by relatively greater pressure on the right side of the operculum; after air has entered the lungs, however, and the pulmonary circulation has been established, the pressure borne by the two surfaces is equal, and hence immobility, and subsequent adhesion of the valve.

Usually within a period of ten to twelve days after birth complete adhesion takes place, and direct communication between the two auricles is thereby entirely and permanently interrupted; but occasionally, although closure of the passage by adhesion has not been effected, by default of nutritive activity in the tissues, or from some other and unknown cause, interauricular communication is not set up, owing, probably, to an equality of pressure existing upon the opposite surfaces of the operculum. Should this equality, however, become deranged from any cause, then intermixture of the venous with the arterial blood may take place, for the first time, it may be, at an advanced period of life.

There is another condition of the valve which may give rise to the passage of blood in the opposite direction, namely, from the left into the right auricle, of which examples will be adduced in the progress of this work; this condition is one, not of defect of adhesion but of development; the upward growth of the valve has fallen short of the superior margin of the opening, and hence patency of the latter, and transit of blood in the direction of least pressure, or from left to right side.

The existence of a third branch of the pulmonary artery, the *ductus arteriosus*, constitutes another distinctive peculiarity of the foetal circulatory system. This branch arises from the root and distal wall of the left division of the pulmonary artery, and in point of size and direction represents the continuation of the

\* *Opus citat.*, p. 412.



parent trunk of that vessel. It is about half an inch in length, passes upwards and to the left, and joins the aorta on its inner surface, at the termination of the arch, that is, a short distance below the point of origin of the left subclavian artery; here the left recurrent laryngeal nerve hooks round it inferiorly, in its ascent to the larynx.

During foetal life, the great bulk of the blood circulating through the trunk of the pulmonary artery passes by the ductus arteriosus into the descending aorta, two minute streamlets only passing by the lateral branches of that vessel into the lungs, whence it is returned to the left auricle by the pulmonary veins. At birth, circulation through the ductus arteriosus is arrested; the vessel begins to shrink, and about the fourth day is completely closed; thenceforward it is represented by an impervious ligament, or fibrous band.

Doctor Alvarenga of Lisbon,\* in a pamphlet of great value, gives the results of his examination of the hearts of infants and children between the ages of one day and twelve years, at the Hospital of St. Joseph, and the Foundling Hospital in that city, with a view to determining, 1st, the time of closing of the foramen ovale; 2nd, that of the ductus arteriosus; 3rd, the relative times of closing of both these structures in the same individual. The total number of hearts examined was 213, and the conclusions at which he has arrived may be summed up as follows, viz.:

1. That the foramen ovale never closes before two and a-half months, and only very rarely indeed before the age of six months, and that it frequently is found open up to the twelfth year.
2. That the arterial canal is never obliterated before the thirtieth day, and very rarely before the sixtieth, and that it is usually closed before the end of the fifth year.
3. That the arterial canal is usually obliterated before the foramen ovale is closed.

The reviewer very justly observes, in relation to Dr. Alvarenga's tables: "Much remains to be cleared up concerning the condition of the foramen ovale between that period (six months)

\* *Brit. and Foreign Med.-Chirurg. Review*, No. lxxxix, January, 1870.

and the twelfth year, before we can have any accurate knowledge of the time of closing of this structure."

Senac erroneously concludes that the closure of the ductus arteriosus is due to "extension," consequent upon the elongation of the aorta by the larger volume of blood circulating through it after the establishment of respiration.

*The Eustachian valve*, at a very early period of intra-uterine existence, was so amply developed as to cover completely the interval between the orifice of the inferior vena cava and the foramen ovale, and form, with the posterior wall of the auricle, a continuation of the cava quite into the foramen, so as effectually to preclude intermixture of the current entering the auricle by the inferior cava, with those from the superior cava and coronary vein. There seems reason to conclude, however, that for a considerable period before birth the valve has been incompetent to effect this object, and that its free margin has become gradually more and more distanced from the annulus ovalis, partly in consequence of the progressive expansion of the auricle, and partly because of the gradual atrophy from disuse, of the valve itself. The closure of the foramen ovale, which takes place at birth, entirely and permanently suspends this function of the Eustachian valve, which seems thenceforward to serve only as a partial "break" to reflux upon the liver in the erect posture of the body.

In the body of the foetus, the internal iliac arteries of the adult, under the name of *hypogastric and umbilical arteries*, and in point of magnitude and direction representing the continuation of the common iliacs, pass upwards in the posterior and the lateral false ligaments of the urinary bladder, to its superior fundus, and thence in the superior or suspensory ligament or fold of peritoneum, to the umbilicus, where they meet and twine around the umbilical vein, and by it are conducted to the placenta. These vessels convey the largest portion of the blood of the common iliac arteries to the placenta, where it undergoes oxidation, and whence it is returned by the umbilical vein to the liver. The division and deligation of the *funis* after birth involves closure of all three vessels, the consequence of which, as regards the hypogastric arteries, is that these vessels become

entirely obliterated as far back as the superior fundus of the bladder, and ultimately by a process of progressive atrophy are converted into fibrous cords, which, resting on the peritoneum, divide the internal inguinal pouches, each, into an inner and an outer portion. From the superior fundus of the bladder backwards to the edge of the great sacro-sciatic notch, where the main branches of the internal iliac are given off, the vessels undergo partial atrophy and diminution of calibre, and under this form they continue through life as the superior posterior vesical arteries.

In the horizontal fissure of the liver, the umbilical vein gives off usually two small branches, which enter and are distributed in the left lobe; and at the junction of the horizontal and transverse fissures it undergoes ultimate division into two branches; of these the larger passes in the transverse fissure towards the right side, to anastomose with the left branch of the *vena portæ* whilst the second and smaller branch, under the name of *ductus venosus*, representing the continuation of the parent trunk, runs upwards and backwards in the horizontal fissure, to open into the left hepatic vein near the junction of that vessel with the vena cava ascendens. During foetal life the intestinal canal is in a state of functional abeyance, or of preparedness to assume at birth its proper function. The portal circulation of the foetus, therefore, represents only the volume of blood circulated in the stomach, intestinal canal, spleen, and pancreas, for the purpose of tissue-nutrition. This, constituting the weaker current, is carried off by that of the right branch of the umbilical vein in the transverse fissure of the liver, and circulated through the medium of the right portal branches in the right lobe of that organ. When, however, alimentary matter is introduced into the stomach after birth, and the digestive viscera assume, for the first time, their special function, "a draught," or afflux of arterial blood towards these organs is the immediate consequence. A full volume of blood now circulates in the vena portæ, whilst, on the other hand, the circulation of the umbilical vein has been entirely arrested by deligation and division of the funis; the current, therefore, now sets in from the portal to the umbilical branches in the liver, and, owing to the attraction of secretion operating upon

the lateral branches, the ductus venosus is entirely drained, and becomes impervious and atrophied, as does likewise the trunk of the umbilical vein from the umbilicus to the transverse fissure of the liver. These vessels are completely obliterated, usually about the fourth day after birth.

Coincidentally with the first act of inspiration a series of important organic changes is initiated in the heart. Consequent upon the introduction of atmospheric air into the lungs, an afflux to these organs through the lateral branches of the pulmonary artery takes place, and the ductus arteriosus being entirely and permanently emptied by the draught so established, undergoes gradual obliteration and wasting. The blood which had now for the first time circulated in large volume through the pulmonary vessels, is poured in equal quantity into the left auricle. The operculum is pressed down from the left side upon the foramen ovale, and becomes gradually agglutinated to its margins, closing the passage, as previously described. The entire mass of the blood entering the right auricle is now carried into the right ventricle, the Eustachian valve undergoes gradual wasting, and the head, neck, and upper extremities, no longer enjoying a monopoly of a comparatively pure circulation, as in foetal life, cease to be developed disproportionately to the rest of the body.

*Physiology of the Heart.* The *rhythm* of the heart means properly its continued alternate contraction and relaxation at determinate intervals of time; but, as commonly understood, a number of concomitant but subsidiary phenomena, manifest to the senses, is included within the meaning of this term. In this broader sense it may be defined as *the regular and uninterrupted succession, and periodic recurrence, in association with the action of the heart, of certain phenomena of sound and of motion, alternating with brief but unequal intervals of silence and repose, and coeval with the life of the animal.*

The entire group of phenomena, positive and negative, constitute an *action* or *pulsation* of the heart, and of these, within the range of health, and in the sitting posture of the body, from 65 to 95 occur within the minute; the former in advanced age, and the latter in childhood. This standard is arbitrary, but at least approximately correct. The difficulty, universally admit-



ted, of drawing a sharp boundary line between the conterminous regions of health and disease, even structurally in the *post-mortem* room, is greatly enhanced when this has to be accomplished in the living body, and by reference to function exclusively. For groups consisting of great numbers an average may be taken, possessing at least approximate accuracy, but for individuals, owing to the many and various shades of difference in individual organization, each, however, potential in its influence upon the rate of cardiac pulsation, a health-rate cannot be fixed save by reference to the collective standard. I am acquainted with persons in reputedly good health, and who enjoy it in the conventional sense, performing all the duties of their station, involving active and constant labour both physical and mental, whose pulse is habitually not above 50 in the minute. The opposite condition, of a permanently quick pulse, is not equally compatible with apparent health. I am not acquainted with an example, in the adult, of a pulse persistently at 90, in which the existence of disease of some kind was not sufficiently manifest.

The phenomena which constitute a pulsation of the heart, and the succession and repetition of which in regular order, and at determinate intervals, constitute its rhythm, enumerated in the order of their succession from an arbitrary starting point in the cycle, are the following, viz., impulse, wave-like movement more or less localized within the region of the precordium, first sound short pause or period of silence, second sound, long pause.

The regular succession, and unfailing repetition of this cycle of phenomena, constitute one of the most remarkable, as it is the most typical manifestation of animal life. Several theories have been propounded in explanation of the alternate contraction and relaxation of the heart, upon which the above-mentioned sensible phenomena depend. These theories may be classified under two heads, namely, those of *direct*, and those of *mediate* stimulation. Both would imply the presence, in the muscular tissue of the heart, of the property of irritability, in virtue of which it is capable of responding to a stimulus by an act of contraction, the duration of which corresponds with that of the stimulus applied.

Haller\* was the first writer who systematized the theory of direct stimulation. According to him, the contractility of the heart is quite independent of nervous influence, and brought into action by the contact of the blood with the internal surface of its several chambers. When contact ceases in any particular chamber by departure of the blood from it, contraction of the walls of that chamber likewise ceases, and thus relaxation supervenes; but, owing to the cyclical character of the circulation, and the existence of an auricle and a ventricle on each side of the heart, the former is of necessity full and in diastole, when the latter is empty and in systole. Thus, an alternate contraction and relaxation is maintained, not only in regard to each chamber, but likewise as between the auricle and the ventricle of the same side of the heart.

Nor is this all; for as the circulation is twofold, systemic and pulmonic, the heart is a duplicate organ, and this double alternation of contraction and relaxation is repeated on both sides.

There seems, *in limine*, a fatal objection to this theory, taken as presented by its illustrious author, namely, that it involves the admission of a pre-existing force to set the blood in motion, ere the chambers of the heart could have been excited to contract by contact with it. Or is it that blood having been primarily formed in the heart itself, the property of irritability, by which its presence there was resented on the occasion of the first, as of all subsequent contractions, was, at the outset, *suddenly* conferred upon the organ? This would seem to be a rather gratuitous and unwarranted assumption. Then, again, is the stimulus of black blood required for the right heart, and of red blood for the left? Bichat† in his experiments on asphyxia, found black blood circulated freely through the left side of the heart. Finally, there is the objection to this theory, that the heart of a cold-blooded animal, *e.g.*, a frog, may continue to pulsate for a considerable period after its removal from the body of the animal, although during the time no blood has been in circulation through it.

Senac‡ advocates the Hallerian doctrine in a modified form.

\* *Opera Minora*, tom. i. p. 155.

† *Traité de Physiologie*, par F. A. Longet, 1869, vol. ii. p. 103.

‡ *Opus citat.*, p. 454.

He maintains that the venæ cavæ and the pulmonary veins contract rhythmically, and propel the blood into the auricles, which, in response to the stimulus of contact, contract upon the blood and force it into the ventricles; and these, resenting its presence, react and urge it into the aorta and pulmonary artery. He adds, these "machines" continue to act "as long as the vital principle resides in the tissue of the brain and of the nerves." These last words imply a radical modification of the doctrine of "inherent irritability" of Haller.

Brücke\* holds that the rhythm of the heart is due to the necessarily intermittent and periodic character of the circulation in the coronary vessels. According to him, the sigmoid valves of the aorta cover the orifices of the coronary arteries, and prevent the entrance of blood into them during the systole of the left ventricle; but not only is the entrance of blood so prevented, but that already in circulation in the walls of the ventricles is mechanically pressed out through the coronary veins, so that the muscular substance is rendered exsanguine *pro tem*. The effect of this depletion is exhibited in relaxation of the walls of the ventricles, by which blood is permitted to circulate freely through them, and the sigmoid valves being depressed at the same moment, the mouths of the coronary arteries are thereby uncovered, and blood is forced into them by the elastic recoil of the walls of the aorta. The immediate consequence of this is contraction of the ventricular walls, which, by rendering them exsanguine as already explained, gives rise, in turn, to relaxation.

There are many patent objections to this theory. It makes no account of the auricles, which, being likewise supplied with blood by the coronary arteries, and being in diastole when the ventricles are in systole, are therefore in a state of receptivity for blood, when, under the conditions of this theory, none can reach them; yet, notwithstanding this deprivation, they contract, instead of relaxing, immediately after ventricular systole. Again, immediately succeeding aortic reaction, when, according to Brücke's theory, blood finds entrance into the coronary arteries, and the ventricles should contract, there is a protracted

\* Longet (*opus citat.*).

period of silence (the long pause), during which the ventricles are undergoing progressive diastole.

Haller and Bertin, moreover, have observed blood to spout from a punctured coronary artery with increased force during ventricular *systole*; and Hyrtl has recently injected the coronary arteries from one of the pulmonary veins.

I have repeatedly examined the root of the aorta, with a view to determining the relationship between the semilunar valves when raised against the walls of the vessel, and the orifices of the coronary arteries. I have usually found the valves to fall short of the mouths of the coronary arteries by several lines. Occasionally I have seen them touch the lower margin of the orifices, *but in no single instance to cover them*.

The experience of Senac\* has been similar. He judiciously observes, that blood in some quantity must enter the coronary arteries during ventricular systole, even though the valves when raised, overlay their mouths; because, in being raised, the valves must force into them a portion of the blood resting on their arterial surfaces, whilst it is being pressed out of the sinuses. Morgagni is of the same opinion.

Brown-Séquard has expressed the opinion† that the circulation of blood through the coronary veins, charged with *carbonic acid*, constitutes the normal stimulus to the contraction of the heart; and M. Lannelongue,‡ availing himself of the doctrine of Brown-Séquard, has essayed to explain the difficulty in the way of the reception of Brücke's theory, arising from the alternating contraction of the auricles and the ventricles. According to his view, immediately the ventricles relax, the re-establishment of the coronary venous circulation in their walls, acting as an excitant to the muscular fibres, brings about a renewed act of systole. During auricular diastole, on the contrary, he assumes that circulation in the walls of these chambers is arrested by collapse and "folding" of them; as the auricles fill, the coronary vessels become patulous, and are disgorged only during auricular systole, because, being attached to the muscular fibres,

\* *Opus citat.*, p. 365.

† *Exper. Researches applied to Physiology and Pathology*, 1853, p. 114.

‡ *Circulation Veineuse des Parois Auriculaires du Cœur*. Thèse de Paris, 1867.



they are held open by their contraction. But the assumption of "collapse" or "folding" of the walls of the auricles is not only gratuitous and unproven, but contradicted by observation and experiment, as will appear further on. The auricles are never empty, because, when disembogued by the divarication of the auriculo-ventricular valves at the moment of cessation of ventricular systole, they are supplied at an equal rate, and with an equal quantity of blood, from the veins opening into them.

Then again, the theory will not hold in reference to the alleged period of disengorgement of the veins, that of auricular systole, because at this period reflux upon the coronary sinus is obviated, either by the closure and adequacy of the valve of Thebesius, or the constriction of the orifices of the vessel by the adjacent contracting fibres. In either case, the orifice would not be free to the *entrance* of blood during auricular systole.

According to the experiments of Erichsen,\* not only the irritability, but likewise the contraction of the heart depends upon the coronary circulation; he has succeeded in arresting the action of the heart by ligaturing the coronary arteries; he found, moreover, that this effect was precipitated by puncturing the coronary veins at the same time, and thus rapidly anæmiating the heart-substance, whilst it was postponed when they were ligatured. Schiff professes to have induced even local paralysis of the heart by tying the artery destined for the supply of that portion of the organ. Whilst, on the contrary, Ludwig, E. Cyon, and Bezold, deny altogether the alleged influence of deligation of the coronary arteries upon the action of the heart; no immediate consequence of this operation is noticeable, and the changes that subsequently ensue in the number and force of the pulsations, are attributed by them to other causes.

The doctrine of the mediate stimulation of the heart involves the physiology of its nerve-supply, or its *innervation*. It has been already shown (p. 35) that the heart derives its nerve-supply from two sources, namely, the pneumogastric and cervical sympathetic. The filaments of these nerves are distributed throughout the substance of the heart, but notably in greatest

\* "On the Influence of the Coronary Circulation on the Action of the Heart :"  
*Lond. Med. Gazette*, 1842.

number on its internal surface, and, in connexion with them minute ganglia have been noticed and described, first by Remak,\* and subsequently by Bowman,† who, however, admits their existence only on the surface of the auricles. From experiments performed by Ludwig‡ and confirmed by Heidenhain§ it would seem that these ganglia are located principally in the vicinity of the auriculo-ventricular septa, for when the heart of a frog was divided transversely a short distance below the auriculo-ventricular septum, the auricles and the attached portion of the ventricles continued to pulsate rhythmically for some time, whilst the inferior fragment, separated by section from the ganglionic centre, ceased to contract immediately. Bidder|| maintains that the coördinating ganglia of the heart are lodged in the inter-auricular septum, and hence the wave-like character of the contractile movement from the auricles towards the ventricles.

To the ganglia situate in the substance of the heart itself, most modern physiologists incline to attribute, as its immediate source, the rhythmical contraction of the organ; but this postulate granted, the precise agency by which the nerve-current emitted from these centres is periodically interrupted, or its influence on the muscle of the heart, whether stimulant or inhibitory, is neutralized at brief and determinate intervals of time, is one of the deepest problems in biology. Mr. Paget,¶ assuming that the nutrition of the heart is alternately quickened and suspended through the agency of these ganglia, proposes to regard its rhythmical contraction as the consequence of "chronometric nutrition." No doubt, the doctrine which regards nutrition as the remote cause or essential condition of all vital action, upon which Mr. Paget's theory rests, is critically incontrovertible, but this only amounts to presenting the difficulty under a new form, without in any degree diminishing or explaining it.

But the nerve-force, whatever may be its character or effect,

\* *Müller's Archiv für. Anat.*, etc., 1844.

† *Physiological Anatomy*, 1856, vol. ii., p. 342.

‡ *Müller's Arch.*, etc., 1848.

§ *Canstatt's Jahresbericht*, etc., 1855.

|| *Müller's Arch.*, 1852.

¶ *Medical Times and Gazette*, January 11, 1859, *Lecture on the Chronometry of Life*, and *Croonian Lecture at Royal Society*, 1857.

which emanates from the intracordial ganglia, is only borrowed from the cerebro-spinal and sympathetic nerve-centres, through the cardiac branches of the pneumogastric and cervical sympathetic nerves. On the assumption, to be presently discussed, that these nerves are reciprocally antagonistic in function, a few eminent physiologists have recently promulgated the doctrine of alternate stimulation and inhibition, as a solution of the problem involved in the periodicity of the heart's movement.

Notwithstanding the emphatic assertion of Haller\* that irritation of the medulla oblongata, spinal cord, or the cardiac nerves, induced no alteration in the action of the heart, physiologists have arrived at the almost unanimous conclusion, that from all three sources a nerve-force emanates, which influences powerfully the rate, the force, and the rhythm of the heart's action. Wilson Philip† was one of the first writers who contributed to the settlement of this question affirmatively, as far as regards the spinal cord. This portion of the nerve-centres of decapitated animals was moistened with alcohol, a solution of opium, and of tobacco, respectively; the first-named agent so applied quickened the action of the heart, whilst the two latter very soon retarded, although at first they accelerated it. He further concluded from his experiments, that the cervical portion of the cord was the most potential in regard to the heart. Prochaska‡ seems to have been the first to assign to the ganglionic nervous system the special function of quickening the contraction of the heart, localizing this function in the cervical, and to a slight extent also, in the superior thoracic ganglia of the sympathetic. Of the independence of this system in regard to its influence over the heart, the examples of amyelencephalic fetuses§ in which the heart continued to pulsate, notwithstanding the absence of brain and spinal cord, afford striking illustration. In the sequel, however, it will appear, that the evidence thus furnished is by no means conclusive as regards the adult mammal.

\* *Dissert. sur l'Irritabilité*, 1756, *traduite de Latin*.

† *An Exper. Inquiry into the Laws of the Vital Functions*: Lon. 1817.

‡ *Opera omnia* (Viennæ, 1800.)

§ Morgagni, *De Sedibus et Causis Morborum*, *Epistola xlviii*.

Legallois,\* in 1811, concluded from a series of experiments, that the spinal cord was the source of the contractility of the heart. These experiments were of a character to show that destruction of the lumbar, dorsal, and cervical portions of the cord respectively, in young rabbits, was followed, within a few minutes, by death from failure of the circulation, despite his efforts to sustain it by means of artificial respiration.

The experiments of Legallois were, within a few years, repeated by Wilson Philip,† and afterwards by Flourens,‡ Longet§ and Brown-Séguard,|| with entirely contradictory results, as regards the absolute and exclusive dominion of the spinal cord over the heart. Bernard, moreover, asserts that the results obtained by Legallois were due to the *pain* involved in the operative procedure, and that the previous administration of chloroform would have entirely obviated them.

That electric irritation of the spinal cord, however, is competent to modify in a very decided manner the force, the rate, and the rhythm of the heart's action, the experiments of Volkmann¶ and Longet,\*\* and those of Wilson Philip, already adverted to, fully establish.

Budge and Ed. Weber,†† in 1845, attributed to certain nerves of the heart the function of restraining its activity. These nerves were named from their alleged function, the *moderator* nerves of the heart.

An influence of this character, under which the heart's action is "slowed," or entirely suspended, the organ remaining in a state of relaxation, may undoubtedly be evoked by stimulation of the medulla oblongata, the pneumogastric nerves, or the abdominal sympathetic; in the two former instances by *direct*, and in the latter by *reflex* irritation.

If the upper part of the medulla oblongata, between the

\* *Ceuvres Complètes avec des Notes de Pariset*, Paris, 1830.

† *Opus citat.*

‡ *Recher. Expér. sur les Propr. et les Fonct. du Syst. Nerv.*, Paris, 1824.

§ *Traité de Physiologie*, 1869, Troisième édition, tom. ii., p. 113.

|| *Exper. Researches*, etc.

¶ *Müller's Archiv.*, 1845.

\*\* *Opus citat.*

†† *Müller's Archiv.*, 1846.



calamus scriptorius and the corpora quadrigemina, be subjected to irritation, the rate of cardiac movement is reduced; and if the irritation be intense, and prolonged beyond a very brief period of time, the heart will cease to contract, and will be found in a state of relaxation. Similar effects will follow from galvanizing the pneumogastric nerves in the neck, and, as shown by Bernstein, the abdominal sympathetic in the frog. If the pneumogastrics be divided in the neck, the pulsations of the heart are promptly increased in frequency, but diminished in force; and stimulation of the medulla oblongata, or of the abdominal sympathetic, will be now without effect upon the heart. If, however, the peripheral portion of the divided pneumogastrics be galvanized, retardation or arrest of the heart's action, according to the intensity of the current, will be the result. It has been already shown that the heart, removed from the body, is capable of sustaining rhythmical action for a lengthened period under favourable circumstances.

It follows, therefore, that the *inhibitory* nerve system of the heart comprises the cerebral origin of the pneumogastric nerves, the pneumogastric nerves themselves, and the ganglia distributed through the heart.

The heart likewise possesses an *excito-motory* system, which consists of the upper part of the spinal cord for centre, and the roots and cardiac branches of the cervical sympathetic for conducting media. If the cervical portion of the spinal cord, or the cervical sympathetic, be galvanized, the heart will pulsate with increased rapidity and force,

Ludwig, however, maintains, that the only portion of the cervical sympathetic which is related to the heart is the inferior cervical ganglion, inasmuch as stimulation of it alone has any effect upon the heart. Dr. Rutherford is of the same opinion; and after discussing very fully the entire subject of the innervation of the heart, this writer summarizes his views in the form of propositions, as follows:

1. The inferior cardiac branches of the vagi are inhibitory of the heart, and in no sense motor, as held by Willis, Lower, Valsalva, Schiff, Moleschott, and Lister.
2. They are not in constant action; activity is the exception.

3. Increase in the rapidity of cardiac movements which follows division of the vagi in the neck, may be due to increase of blood-pressure merely, and this latter to contraction of the gastric blood vessels (owing to suspension of the reflex "depressory" function of the vagi upon the abdominal vessels.
4. The contractile coats of the entire vascular system are supplied with *motor* and *inhibitory* nerves.
5. During digestion the blood vessels of the stomach are dilated, chiefly by vaso-inhibitory action of the vagi upon the splanchnic nerves.\*

These two systems, the inhibitory, and the excito-motory, are connected with one another *centrally* through the spinal cord and medulla oblongata, and *peripherally* through the cardiac plexuses and the ganglia distributed through the substance of the heart.

The necessity for the existence of two opposing and mutually countervailing systems of nerves in the heart, would seem to be involved in the *promptitude* with which, in carrying on the circulation, it is required to contract and relax alternately.

It is alleged that the rapid and continued succession of powerful motory nerve-impulses to which the heart is subjected, would, as in the case of voluntary muscle similarly treated, give rise to persistent spasm, or tetanic contraction of the organ, a condition incompatible with the maintenance of the circulation. An "inhibitory" nerve system, however, which operates, not in the Hunterian sense, by inducing a state of "active relaxation" of the heart, but by neutralizing or suspending, *pro tem*, the motor influence directed upon it from and through the excito-motory system, and which, in turn, either by temporary suspension or relative diminution in the force of its nerve-current, is controlled or overpowered by that system, is manifestly competent to supply the conditions necessary for the maintenance of the circulation, under the special circumstances indicated.

Longet † asserts that the necessity for temporary "inhibition" of the heart has reference to the repose and nutrition of the

\* *Journal of Anatomy and Physiology*, No. iv., May, 1869.

† *Opus citat.*, p. 120.

organ. This is manifestly true ; but a necessity no less urgent, requiring the temporary and alternate suspension of contraction in each of the chambers of the heart, has reference to the maintenance of the general circulation. It is manifest that the distension of each of those chambers with a fresh charge of blood, preparatory to a new act of contraction, implies an antecedent state of relaxation of its walls.

Division of the pneumogastric nerves in the neck gives rise to derangement of the normal ratio between respiration and cardiac pulsation. The former is "slowed" by one half, whilst the latter is doubled according to Bernard. Inspiration is full and deep, yet the blood is imperfectly arterialized, and notwithstanding the excited action of the heart, arterial tension is notably reduced.

Bernard remarks, as a singular anomaly, that in the dog, the pulse, which is normally irregular, is rendered perfectly regular by section of the pneumogastrics. In the frog, on the other hand, Brown-Séquard alleges that division of these nerves has no influence whatever upon respiration or cardiac pulsation.

Galvanization of the pneumogastrics, or of the peripheral extremity of the divided nerves, will cause arrest of the heart in a state of diastole. For this purpose a strong current is necessary ; but if too powerful, or long continued, it will have the opposite effect of accelerating the action of the heart, like section or ligature of the nerves. In this case, likewise, the force of the heart will be lessened, as evidenced by diminished arterial tension. When the current has been stopped, the pulsations return gradually to their normal state. Schiff asserts that galvanization of a single nerve, if sufficiently energetic, will suffice to produce the effects above mentioned.

The hypothesis of "nervous exhaustion" has been adduced, singularly enough, by different physiologists, to explain both the quickening and the arrest of the heart's pulsations, which result respectively from section and galvanization of the pneumogastric nerves in the neck. It is manifest that one of these doctrines must be erroneous. Schiff, Spiegelberg, G. Valentin, Jos. Lister, and Moleschott, hold that the arrest of the heart's action is due to this cause ; but this doctrine would imply that

the pneumogastrics act as motor nerves upon the heart, a position utterly untenable, because irreconcilable with the admitted fact, that whilst a moderately strong current stops the action of the heart, a very strong, or a moderate but prolonged current, will induce rapid but feeble pulsation. The last-mentioned result is presumably due to exhaustion, but if so, the former, which is precisely the reverse, cannot be in any degree attributable to the same cause.

Curara poison previously administered, by annihilating the excitability of the heart, will neutralize the effect of a galvanic current applied to the pneumogastric nerves. Digitalis in large doses will arrest the heart in a state of diastole; its effect upon the heart is therefore similar to that of galvanization of the pneumogastric or its central nucleus; and that it acts upon the heart through the same channel was proved by the experiment of Traube, which showed that it fails to act where the pneumogastrics have been previously divided.

Waller\* alleges that the "inhibitory" property of the pneumogastric nerves is derived from the spinal accessory. He found that when, after destruction of the spinal accessory of one side, the divided roots had undergone fatty degeneration, galvanization of the corresponding pneumogastric failed to cause arrest of the heart, and Longet† has shown experimentally that a motor nerve, detached from the cerebro-spinal centre, ceases to be excitable after the fourth day.

The question of the innervation of the heart was, in 1863, still in a state of uncertainty, to the extent that it was not settled, on the one hand, whether the heart is entirely independent of the spinal cord, and on the other, whether the central nervous system can influence the heart, save through the pneumogastric nerves; the experiments and conclusions previously adverted to, notwithstanding.

In that year (1863) Professor Bezold concluded from the following experiment, that he had proved the existence in the spinal cord of an excito-motory centre of the heart, which was

\* *Expérience sur les Nerfs Pneumogastriques et Accessoires de Willis* (Gazette Médicale de Paris, 1856).

† *Recherches Experimen.* (Examinateur Med. 1841).



capable, not only of increasing the number of its pulsations, but likewise of augmenting considerably the mean pressure of the blood; section of the cord at the level of the atlas in rabbits reduced the pressure in the large arteries, and slowed the heart. Irritation of the cord below the level of section increased both the vascular pressure and the number of cardiac pulsations. These results he attributed exclusively to diminution or augmentation of force of contraction of the heart. MM. Ludwig and Thiry found the same results to follow these experiments, after destroying all the nerves of the heart by the galvano-caustic method, and that pressure on the abdominal aorta caused equal acceleration of heart and increase of vascular pressure; they arrived at the seemingly warrantable conclusion, that the results obtained were due, in the one case to paralysis, and in the other to irritation of the vaso-motor nerves, which, by causing diminution or increase of vascular resistance to the heart, reduced the pressure and rate of pulsation, or increased both.

Ludwig and E. Cyon, by their conjoint experiments, have established; firstly, that the cardiac nerve arising by two roots from the pneumogastric and the superior laryngeal, is a sentient nerve of the heart, conferring on it the power of regulating vascular pressure throughout the body, by inducing, through reflex influence, paresis of the vaso-motor nerves, and so temporary paralysis of the walls of the blood-vessels: to this nerve they therefore propose to give the name of *depressor nerve*. And secondly, that the splanchnic nerves are the principal vascular nerves of the body, section of them reducing pressure in the carotids to a minimum, and irritation of the distal ends of the divided nerves doubling it.

In the hope of excluding, by section of the splanchnics, all vascular changes during irritation of the spinal cord, the brothers Cyon\* performed the following experiments: Rabbits were poisoned with curara, and artificial respiration was kept up; the pneumogastric, the sympathetic, and the "depressor" nerves were next divided in the neck, and then the number of beats of the

\* Sur L'Innervation du Cœur par MM. E. et M. Cyon, de Saint Pétersbourg (note transmise par M. Cl. Bernard) *Comptes Rendus de L'Academie des Sciences*, lxiv., Mars, 1867.

heart was registered, and by means of the manometer of Ludwig the blood-pressure in the carotids was determined, *before, during, and after* electric irritation of the spinal cord, separated from the brain at the level of the atlas. After having ascertained a considerable augmentation of blood-pressure, and an increase in the number of cardiac pulsations, consequent upon irritation of the spinal cord, they divided the two splanchnics below the diaphragm, and quickly the blood-pressure, and the number of pulsations of the heart, descended even below the standard attained after division of the spinal cord *only*. Irritation of the cord, after division of the splanchnic nerves, produced again a considerable acceleration of the action of the heart, *but no alteration of blood-pressure*. From these experiments it may be inferred that spinal irritation causes accelerated pulsation of the heart through the cardiac nerves; and increased vascular pressure through the splanchnics, by means of the vascular resistance induced.

The "depressor" nerve was divided, and the peripheral extremity galvanized without result. The central end of the divided nerve was next subjected to galvanic irritation; the animal immediately gave evidence of pain, and there was a fall of five to six centimetres in arterial tension. These phenomena ceased immediately the current was suspended. If the cardiac nerves were all previously divided, the above-mentioned consequences nevertheless followed; but if the splanchnics were divided they promptly ceased to be manifested.

These experiments prove that the "depressor" nerves are centripetal; and that they exercise their reflex influence, not immediately through the cardiac nerves upon the heart, but on the blood-vessels of the body through the vaso-motor system, of which the splanchnics are the great centre; and finally, that this influence is of a paralyzing or paresic character, causing relaxation of the vascular walls, dilation of the vessels, and consequent subsidence of arterial tension.

All the branches which the heart receives from the spinal cord, through the medium of the lower cervical and upper dorsal ganglia, were then extirpated in other rabbits; the splanchnics were divided, and the cord irritated as before, but no change occurred, either in the number of cardiac pulsations, or in the

mean intra-vascular pressure; thus confirming the preceding inference. It was noted, however, that when the irritation was protracted, an elevation of two to three millimetres in the vascular pressure took place, which was attributed, with some probability, to irritation of the vascular nerves arising lower down than the splanchnics. It was found, moreover, that extirpation of the spinal roots of the cardiac nerves *alone* produced no change either in the number or value of the contractions of the heart. From this the experimenters conclude, I think unwarrantably, that "these nerves do not act in a continuous manner." In my opinion it rather shows, that whilst these nerves constitute the ordinary channels for the conduction of positive irritation from the spinal cord to the heart, the latter organ may discharge, temporarily at least, its ordinary functions independently of them, obtaining its supply of nerve-force from its intrinsic ganglia.

Electric irritation of the third branch of the inferior cervical ganglion provoked, in rabbits, an acceleration of the beats of the heart, and a diminution of their length; and the first and second branches of the same ganglion were found to be sentient nerves of the heart, and to constitute a continuation of the "depressor" nerve.

Irritation of the fourth branch of the same ganglion, which passes in front of the subclavian artery, and forms with the fifth the annulus of Vieussens, produced a slight elevation of blood-pressure without changing the number of the pulsations.

The *direct* irritation of these cardiac nerves, in dogs and rabbits, produces less acceleration of the heart's action than *indirect* irritation through the spinal cord, because, in the latter case, *all* the cardiac nerves are simultaneously irritated.

The third branches of the inferior cervical ganglia, they propose, therefore, to designate the *accelerator* nerves of the heart. They are not ordinary motor nerves terminating in the muscle of the heart, because irritation of them will not give rise to tetanic contraction of that organ, nor increase the sum of work done by it, as shown by the fact determined by means of the manometer, that whilst the number of pulsations was increased, their force was lessened; and also because curara will not para-

lyse them. Neither are they the nerves which act upon the nutrient vessels of the heart, because occlusion of these vessels will not give rise to acceleration of the heart's action.

The conclusions arrived at in regard to the termination and special function of the "accelerator" nerves are, that they end in the ganglia of the heart; that they are subject to the reflex influence of a great number of the sentient nerves of the body, but especially those distributed on the internal surface of the heart; that their action consists in effecting a change of the division *in time* of the heart's work; and finally, that they are in antagonism to the pneumogastrics in this sense, that whilst irritation of the latter "slows" the heart but increases the force of its contractions, irritation of the "accelerator" nerves increases the number of the pulsations, but diminishes their force.

Brown-Séguard\* is of opinion that the pneumogastrics are the vaso-motor nerves of the heart, and that their "inhibitory" influence upon that organ is the result of contraction of the coronary vessels, and temporary anæmia of the walls of the heart. But this view of the function of the cardiac branches of the pneumogastrics would imply, that under their active influence the walls of the heart are in a state of anæmia, which is not the case; and that, as in the experiments of Erichsen and Schiff, of ligaturing the coronary arteries, the relaxation of the heart would be slow and gradual, and the arrest of its action complete only after an interval of several seconds consecutive to the operation; not sudden and prompt as in the experiment of galvanizing the pneumogastric.

That the ganglia of the heart possess, and may exercise for a considerable period independently of the nerve centres, a power of influencing the movements of the heart; and that this power is regulated by, or dependent upon, the blood-supply furnished by the coronary arteries, seems proved by the following experiment of Bezold.† When he closed the trunks of the coronary arteries or several of their branches, by means of forceps, after having divided the spinal cord, the vagi, and the sympathetic nerves in the neck, he observed that during the first ten or

\* *Gazette Med. de Paris*, 1853.

† Quoted by Eulénberg, *Medical Times and Gazette* of 23rd April, 1870, p. 433.



fifteen seconds no perceptible change took place; then the heart's beats became slow and irregular, half or three quarters of a minute intervening; and after a minute or a minute and a-half the ventricles ceased to act. But when the obstruction was removed, pulsation began again, and soon became perfectly regular.

From what has preceded, it will be evident that the special and distinctive functions of the several nerves of the heart have not been yet clearly determined.

After an excellent *résumé* of this subject, Longet\* remarks, "We admit, nevertheless, that new experimental researches are necessary, in order to determine the precise laws of the innervation of the heart."

Convinced of the truth of this statement, and no less, of the importance of the subject in regard to the localization and treatment of certain symptoms, not only of cardiac, but of febrile disease, I determined, in conjunction with my young friend Dr. Furlong, whose information and special aptitude for investigations of this kind qualify him in a high degree for the task, to undertake a new and independent series of experiments, with the view of still further elucidating it, and of endeavouring especially to settle the question of the precise and special role of the individual nerves in the physiology of the heart. I regret to say, however, that after Dr. Furlong had made all the preliminary arrangements, devised and constructed instruments, procured a supply of curara, and received from the Royal Irish Academy a grant in furtherance of his experimental inquiry,† the investigation was abruptly brought to an end by his having been called to an extensive practice in the country,‡ which absorbed his whole time. I thus, to my great regret, found myself without a collaborateur, and unable to command the time necessary to pursue the investigation single handed. I sincerely hope that some physiologist, younger, more vigorous, and in other respects better qualified for the task than I am, may undertake and accomplish it.

\* *Opus citat.*, p. 124, foot note.

† A preliminary Report, together with the instrument devised for the purpose of carrying on artificial respiration, was submitted to the Royal Irish Academy in the session 1872-3.—*Vide Report of Proceedings.*

‡ Enniscorthy.

Having discussed somewhat fully the anatomy of the heart generally, and its nervous endowment in particular, I may now enter upon the consideration of its physiology. A clear conception of the mechanism of the heart; of the agency by which its movements, and those of its several parts, are initiated and controlled; of the coincidence or the asynchronism of its many sensible phenomena; and of the character, quality, and duration of each of these in the state of health, is absolutely necessary as a first step in the acquisition of a knowledge of the deviations which indicate derangement of its function; the source whence they proceed; the seat of their origin; the cause to which they are due; their diagnostic and prognostic significance; and the therapeutic indications arising therefrom. I am guilty of no exaggeration in asserting that upon this subject, even in the minds of otherwise accomplished physicians, the utmost confusion has existed, and still, but in a much less degree, exists.

The substitution of accurate knowledge for hasty assumption and adventurous hypothesis, upon a matter of so much importance, should, therefore, be the first object of any one undertaking to produce a treatise on the Diseases of the Heart, on a level with the present advanced knowledge of its physiology.

It has seemed to me that much useful information may be afforded by presenting, in a synoptical form, the principal hypotheses hitherto propounded in regard to the rhythm of the heart, and the grouping or succession of its several phenomena. But as these are presented in a cycle, which, throughout life, is being constantly repeated, without material deviation in health, but exhibiting in disease various alterations of corresponding significance, the diagrammatic scheme of a circle seemed best adapted to the object in view.

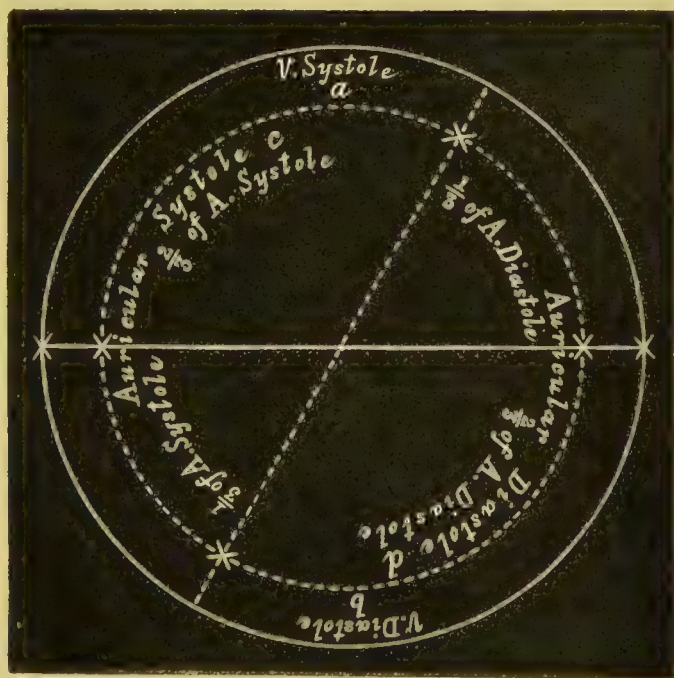
In the construction of this scheme the cardinal point to be attended to was the contraction of the ventricles, because with it were associated in the minds of all writers on the subject, with only three notable exceptions, and upon it depend, the leading phenomena of impulse and first sound. The relaxation of the ventricles possesses only a reflected value, and constitutes the second point of importance to be attended to.

But these two principal phenomena were found to cover the

entire period of a cardiac cycle, and therefore all the minor phenomena were readily grouped in two great divisions under them.

Lancisi (Fig. I.) divided the cycle into two equal portions, namely, ventricular systole, and ventricular diastole. Within the former were grouped the impulse of the heart, the first sound, the two posterior thirds of the auricular systole (coinciding with the two anterior thirds of ventricular systole), closure of the auriculo-ventricular, and opening of the arterial valves, escape of blood from the ventricles into the aorta and pulmonary artery, and distension of these vessels. Within the period of ventricular diastole occurred the second sound, the anterior third of auricular systole (coinciding with the terminal portion of ventricular diastole), opening of auriculo-ventricular, and closure of arterial valves, and reaction of the aorta and pulmonary artery. The subjoined diagram will render this intelligible and easy to remember.

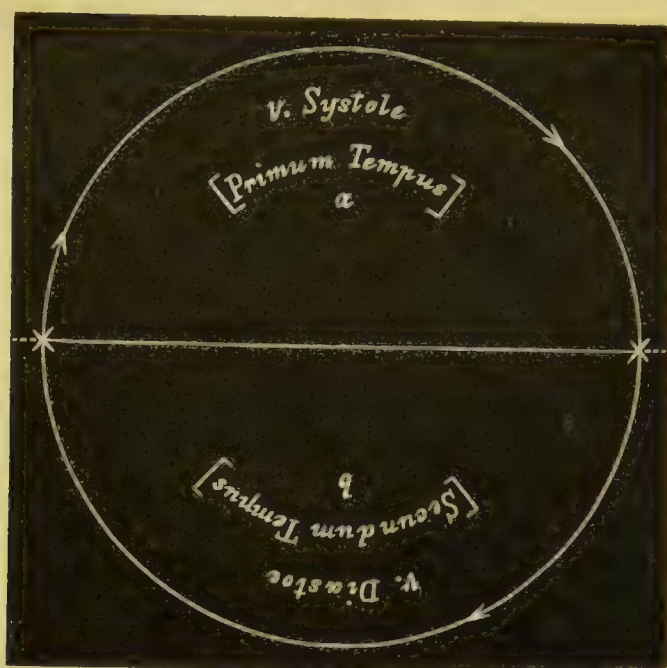
FIG. I.



a	*	...	...	...	*	Ventricular systole.
b	*	...	...	...	*	Ventricular diastole.
c	*	...	*	...	*	Auricular systole.
d	*	...	*	...	*	Auricular diastole.

The manifest objection to this scheme is that it assumes,

FIG. II.



- a \* ... .. \* Ventricular systole (*primum tempus*).  
 b \* ... .. \* Ventricular diastole (*secundum tempus*).

contrary to all mechanical principles, and to observed facts, that the contraction and the dilatation of the auricles and of the ventricles respectively, are in greatest part simultaneous. It may be, therefore, set aside without further discussion; but historically it is worthy of being recorded as evidence of the importance with which, even at that early period,\* a concrete knowledge of the rhythm of the heart was invested in the mind of a great physiologist and physician.

Haller (Fig. II.) divided the entire cycle into two equal portions, corresponding respectively to what he designates *primum tempus*, and *secundum tempus*. Within the former period occur the following phenomena, viz.: contraction of the ventricles, impulse, first sound, displacement of anterior or larger segments of auriculo-ventricular valves from arterial orifices, and closure of these valves over the corresponding orifices, with repulsion into auricles of a "cone of blood," raising of arterial valves, and distension of aorta, pulmonary artery, and auricles.

\* First decade of seventeenth century.

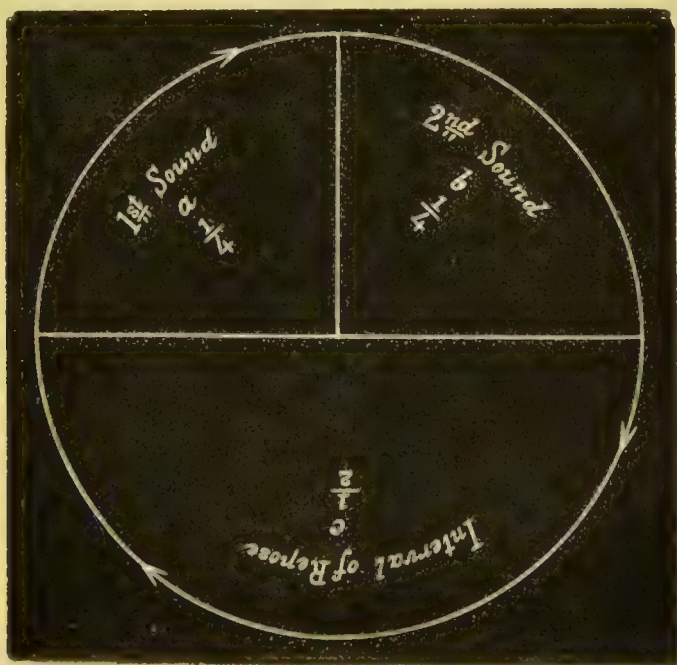


During the latter period (*secundum tempus*) occur the relaxation of the ventricles, and entrance of blood from the auricles, overlapping of arterial orifices by anterior segments of auriculo-ventricular valves, reaction of aorta and pulmonary artery, closure of arterial valves, second sound, and contraction of auricles.

This sketch is in greatest part correct, so far as it extends; the most notable exception being, that the auriculo-ventricular valves are floated athwart the corresponding orifices by the blood entering from the auricles, instead of being forcibly raised, and lifting a column of blood into the auricles, under the contraction of the ventricles, as implied by Haller. There is, moreover, a want of fulness of detail, and a total omission of several features essential to a complete scheme, such as an apportionment of time.

Laennec regarded the sounds as the principal phenomena, attributing the first sound to the contraction of the ventricles, and the second sound to that of the auricles. The error implied in this assumption, as well as that of the relative apportionment of the entire period of the cycle, exhibited in the next figure (Fig. III.), requires no explanation.

FIG. III.



- a First sound (Ventricular systole) =  $\frac{1}{4}$  of cycle.
- b Second sound (Auricular systole) =  $\frac{1}{4}$  of cycle.
- c Interval of repose of entire organ =  $\frac{1}{2}$  of cycle.

He held that there was no "interval of repose" between the first and second sound; but herein he was in error, as he likewise was in the assumption, that every twenty-four hours the ventricles enjoyed twelve, and the auricles eighteen hours of repose. If, by diastole, is meant ventricular repose, then the ventricles actually enjoy sixteen hours of such out of every twenty-four. But diastole is not repose or immotion; it is relaxation and passive motion, and not entirely exempt from tissue-waste. The auricles, as will be hereafter pointed out, are in action during the entire period of ventricular diastole, *i.e.*, during two-thirds of the period occupied by a cardiac cycle; but in vigorous action only in the terminal portion of this period. Their state of relaxation corresponds to ventricular systole, *i.e.*, one-third of the cycle; but during ventricular systole the auricles are in a state of extreme distension, which cannot be regarded as repose. With the first sound, according to Laennec, the impulse of the heart and the radial pulse coincide.

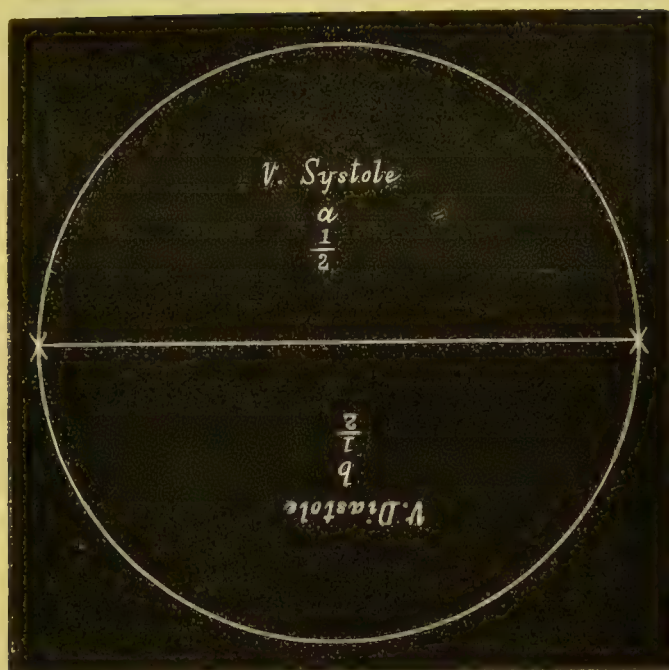
Bouillaud (Fig. IV.) divided the period of cardiac action into two equal parts, viz., ventricular systole, and ventricular diastole.

During the systole occur, the impulse of the apex; first sound; recession of the body of the ventricles; pulsation of adjacent arteries; repose of auricles and suction of blood into them; contraction of papillary muscles and consequent closure of auriculo-ventricular valves; and efflux of blood into the aorta and pulmonary artery.

Within the period of diastole occur, the retrocession of the apex from the chest-wall; the second sound; slight retraction of auricles; repose and partial distension of ventricles; vermicular contraction of auricles, commencing in appendices and ending in ventricles, where it merges in ventricular systole; reaction of aorta and pulmonary artery, and consequent closure of sigmoid valves, after being drawn towards base by ventricular diastole.

In reference to this scheme I will merely observe, that the retreat of the body of the ventricles from the anterior thoracic wall after the impulse, takes place, not within the period of ventricular systole, but during the subsequent diastole; and that no notice is taken of the energetic contraction of the auricles, which

FIG. IV.



*a* Ventricular systole =  $\frac{1}{2}$  cycle.

*b* Ventricular diastole =  $\frac{1}{2}$  cycle.

occurs immediately before the contraction of the ventricles, upon which the latter depends, and to which, as will be shown in the sequel, the pathognomonic murmur of mitral constriction is mainly due.

The Dublin Committee of the British Association (1835) divided the entire period of the heart's action into three unequal portions (Fig. V.); one-half corresponding to the ventricular systole, the impulse, and the first sound, which are all of equal length; one-eighth belongs to the second sound; and three-eighths to the subsequent pause, during which diastole of the ventricles, and systole of the auricular appendices occur. The first sound is caused by *bruit musculaire*, and rushing of blood over the rough surface of the ventricles; it is intensified by impulse against the wall of the chest. The second sound is due exclusively to closure and tension of the sigmoid valves. This scheme, though possessing much merit, and being decidedly the best up to that date produced, because based upon direct observation and ex-

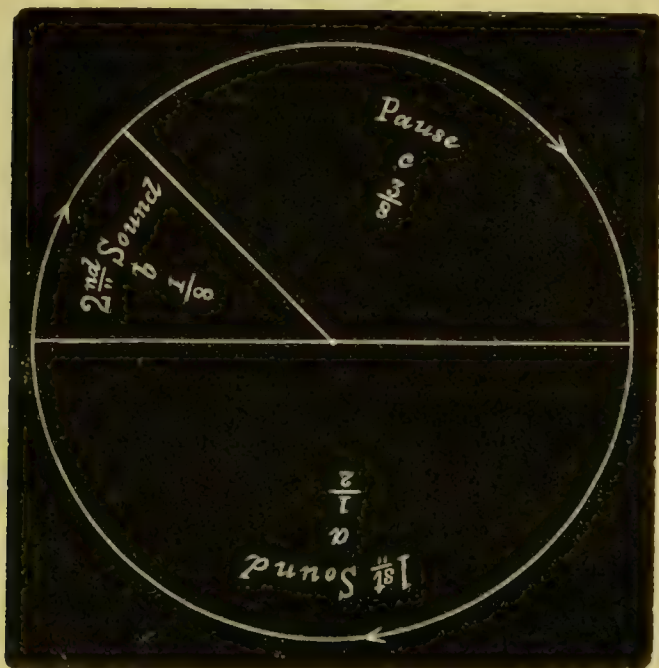


periment, was erroneous in assuming that the diastole of the auricles does not occur till after the lapse of an appreciable interval subsequent to the systole of the ventricles, whereas it actually coincides with the latter. It was also at fault in excluding the second sound from the period of ventricular diastole; and in representing the auricular appendices only as contracting during the pause. The division of time was likewise incorrect; and the impulse and first sound were both erroneously assumed to be equal in length to the systole of the ventricles.

The following summary of the conclusions arrived at by the Dublin Committee are eminently worthy of attention, as indicating the great acumen of the observers constituting that distinguished body, and as presenting the least incorrect view of the action of the heart propounded up to that date:

1. "In warm-blooded animals the systole of the ventricles follows immediately that of the auricular appendices.
2. "During the systole of the ventricles the auricles are distended by blood from the venous trunks.

FIG. V.



- a* First sound (ventricular systole and impulse) . . . . . =  $\frac{1}{2}$  cycle.  
*b* Second sound . . . . . =  $\frac{1}{3}$  cycle.  
*c* Pause (ventricular diastole, and systole of auricular appendices) =  $\frac{2}{3}$  cycle.



3. "When the systole has ended, the ventricles become relaxed and flaccid, and blood passes rapidly, but not with force, from the auricles into their cavities.
4. "The auricles are never emptied of their blood, and contract but little on their contents, an active contraction being observable only in their appendices.
5. "If the interval between two successive beats of the heart be regarded as divided into four equal parts, two of these may be allotted to the duration of the ventricular systole, something less than one to the interval between the termination of the ventricular systole, and the beginning of the diastole of the appendices, during which interval little motion is observed in the auricles; and the remainder to the diastole, and systole of the auricular appendices.
6. "The ventricles in their systole approach the front of the thorax, and by their contact and pressure against it, produce the impulse or beat of the heart."

Corrigan divided the cycle into three unequal parts (Fig. VI.), regarding the sounds as the principal events, after the example of the Dublin Committee. The first sound occupied one half the cycle, and coincided with the impulse of the heart, the contraction of the auricles, and dilatation of the ventricles. The second sound extended over one-fourth of the cycle, and coincided with the contraction of the ventricles, the distension of the aorta and pulmonary artery, and the radial pulse. The pause engaged likewise one-fourth of the cycle, and corresponded to the distension of the auricles and relaxation of the ventricles.

This scheme is eminently heterodox, and has been recently repudiated by its eminent author, who, on witnessing some conclusive experiments by M. Chauveau,\* is understood to have announced his conversion.

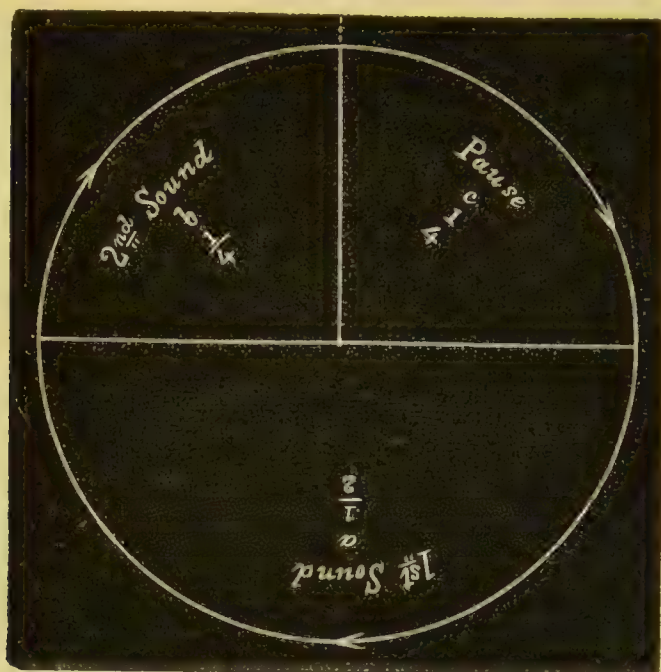
Long anterior, however, to the date of Chauveau's exposition, he had practically abandoned it, and conformed to the orthodox view, as is well known to his professional brethren in Dublin. Nevertheless, as no formal retractation has been published, it is necessary to state briefly the objections to this scheme.

The first sound was made to coincide with the contraction of the auricles, and dilatation of the ventricles. The second sound

\* *Dublin Hospital Gazette* : November 1, 1860.

was represented as coinciding with the contraction of the ventricles and the radial pulse; and from the pause was excluded

FIG. VI.



- a* First sound (contract. of auricles, dilatation of ventricles, and impulse) =  $\frac{1}{4}$  cycle.  
*b* Second sound (contract. of ventricles, and radial pulse) ... .. =  $\frac{1}{4}$  cycle.  
*c* Pause (distension of auricles, and relaxation of ventricles)... .. =  $\frac{1}{4}$  cycle.

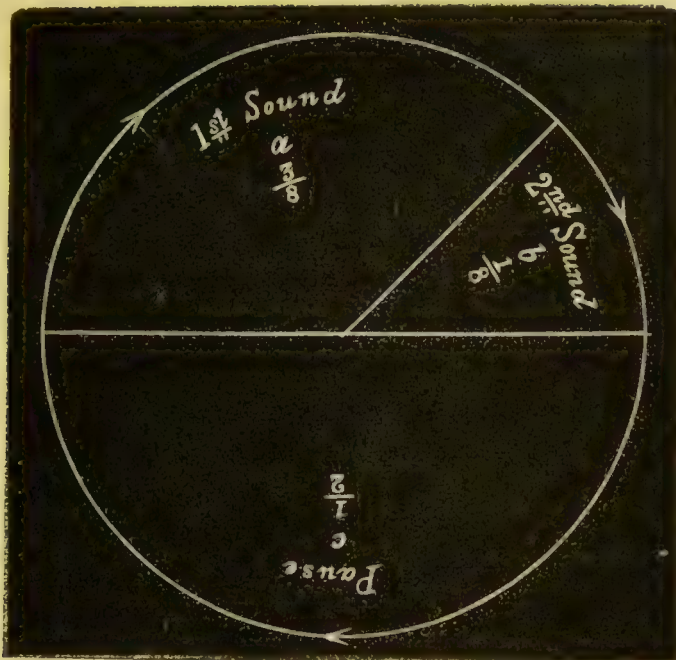
the contraction of the auricles. The radial pulse could never, in this view, be anterior to the second sound, since ventricular systole on which it depends, coincides with that sound; yet it is well known to be normally antecedent to it. M. Beau takes a similar view as to the cause of the sounds of the heart, and is, therefore, likewise amenable to the foregoing strictures.

Corrigan adverted to "a mistake made by Laennec, who stated that the ventricles contracted before the auricles. It was now generally agreed that Laennec was wrong."\* I fail to perceive the force of this remark, since any one point of a circle may occupy a place before or after any other point in it, according to the point of departure selected.

Carlile divided the cycle into three parts (Fig. VII.); the first, occupying three-eighths, corresponds to the first sound, the sys-

\* British Association, 1835.

FIG. VII.



- a* First sound (v. systole) ... .. =  $\frac{3}{8}$  cycle.  
*b* Second sound ... .. =  $\frac{1}{8}$  cycle.  
*c* Pause ... .. =  $\frac{1}{2}$  cycle.

tole of the ventricles and closure of the auriculo-ventricular valves, impulse of the heart, distension of the aorta and pulmonary artery, and the diastole of the auricles. The second part represents the second sound, and is one-eighth of the whole cycle; it corresponds likewise to the reaction of the aorta and pulmonary artery, closure of the sigmoid valves, relaxation of the ventricles, opening of the auriculo-ventricular valves, and entrance of some blood from the auricles. The third part, amounting to one-half of the cycle, represents the pause, and corresponds to the systole of the auricles, which commences at the entrance of the venæ cavæ and ends at the appendix; the diastole of the ventricles, and entrance of the principal volume of blood from the auricles.

This was the most truthful and accurate sketch given up to the date of its publication; still, several objections apply to it. The time is incorrectly apportioned, the sounds are made the principal events, and the systole of the auricles is placed at the commencement, instead of the termination of the long pause.

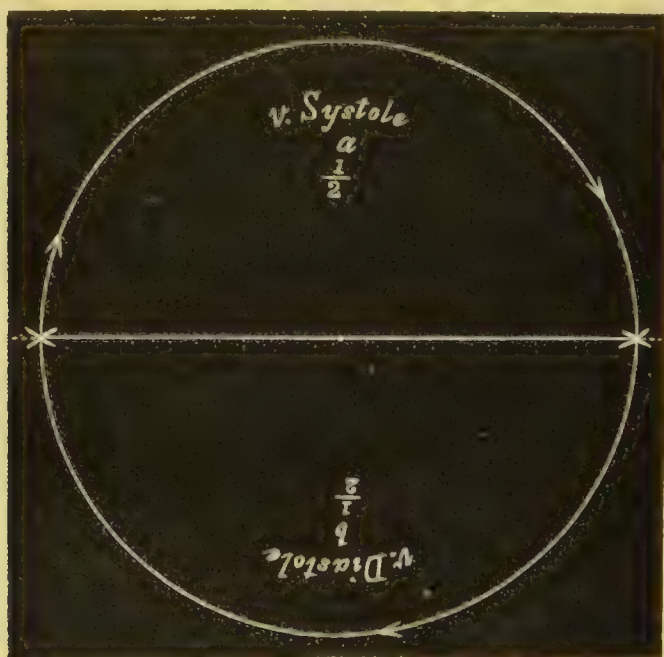
He attributed the first sound to the rushing of blood through the aortic and pulmonic orifices.

Hope implies a division of the cycle into two equal parts (Fig. VIII.) The first portion is appropriated by the ventricular systolic phenomena, viz.: first sound, impulse, and passage of blood into the great arterial trunks. To the last-mentioned phenomenon he attributes the first sound. The second period is occupied by the ventricular diastolic phenomena, viz.: the second sound, and the passage of blood from the auricles into the ventricles, to which that sound is due.

This is a most defective scheme, and erroneous *quoad* the causes to which the sounds of the heart are attributed.

Gendrin furnishes a scheme of great complexity (Fig. IX.), but possessing some special merits. It is, however, in many particulars erroneous. In it the cycle is divided into six parts: viz., *systole*, equal to one-fourth of cycle; *perisystole*, *prediastole*, and *diastole*, together corresponding to one-fourth; *peridiastole*, and *présystole*, together equal to one-half. Within the

FIG. VIII.

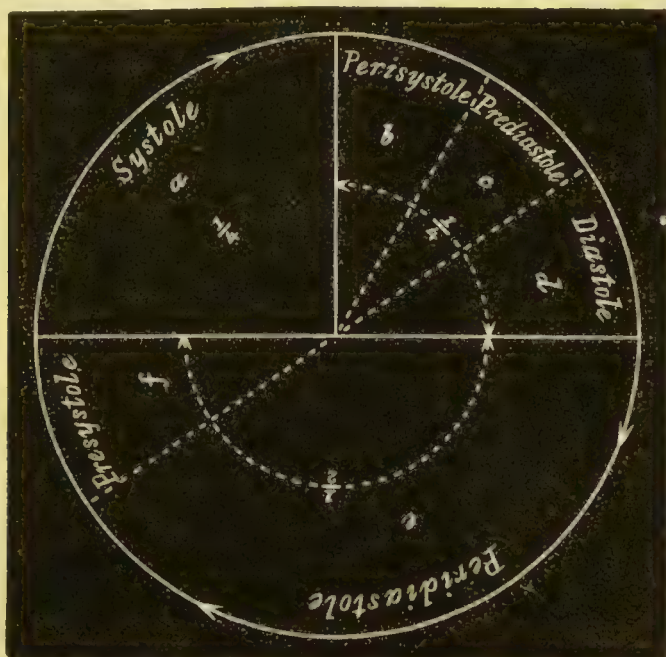


- a* Ventricular systole, etc. ... .. =  $\frac{1}{2}$  cycle.  
*b* Ventricular diastole, etc. ... .. =  $\frac{1}{2}$  cycle.



period of systole occur, the contraction of the ventricles, first sound, impulse, efflux from ventricles into great arteries, initial diastole of latter, and vibration of corpuscles of blood. The perisystolic phenomena are, incipient relaxation of ventricles, and closure of arterial valves. In the prediastole the preceding phenomena are continued.

FIG. IX.



a	Systole	...	...	...	...	=	$\frac{1}{4}$	cycle.
b	Perisystole	}	...	...	...	=	$\frac{1}{4}$	cycle.
c	Prediastole							
d	Diastole							
e	Peridiastole	}	...	...	...	=	$\frac{1}{2}$	cycle.
f	Presystole							

The diastole comprises the relaxation of the ventricles and auricles; opening of auriculo-ventricular valves, and entrance of blood from great veins into auricles, and from auricles into ventricles; recoil of this blood from apex against base and closed arterial valves; second sound.

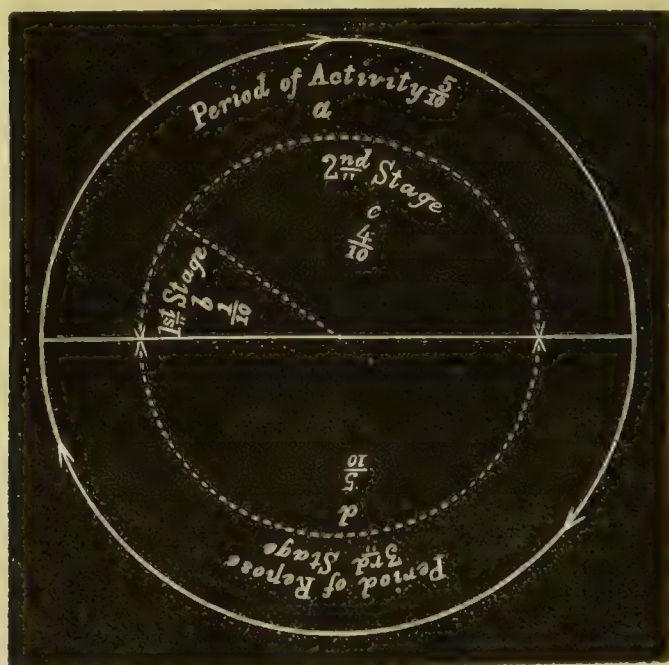
Peridiastolic phenomena are, complete relaxation of auricles and ventricles, and free entrance of blood from great veins into auricles, and from auricles into ventricles; and during presystole, contraction of the auricles and rapid entrance of blood into ventricles take place.

The first sound Gendrin attributes to the vibration of the corpuscles of the blood; and the second sound, to the recoil of the blood first entering the ventricles, from the apex, against the base of the heart and the closed arterial valves. He holds that the closure of the arterial valves precedes the second sound.

The three events, however, are really simultaneous; namely, closure of the sigmoid valves, the second sound, and the first entrance of blood into the ventricles; and, as is now well established, the second sound is caused by the abrupt closure and tension of the sigmoid valves. He alleges that the second sound exists after the sigmoid valves have been destroyed. This is an error, as proved by experiment on living animals, and likewise by the total absence of second sound in the aorta when the sigmoid valves are so much disorganized as to be entirely incompetent.

M. A. Luton,\* copying the scheme of Marey and Chauveau

FIG. X.



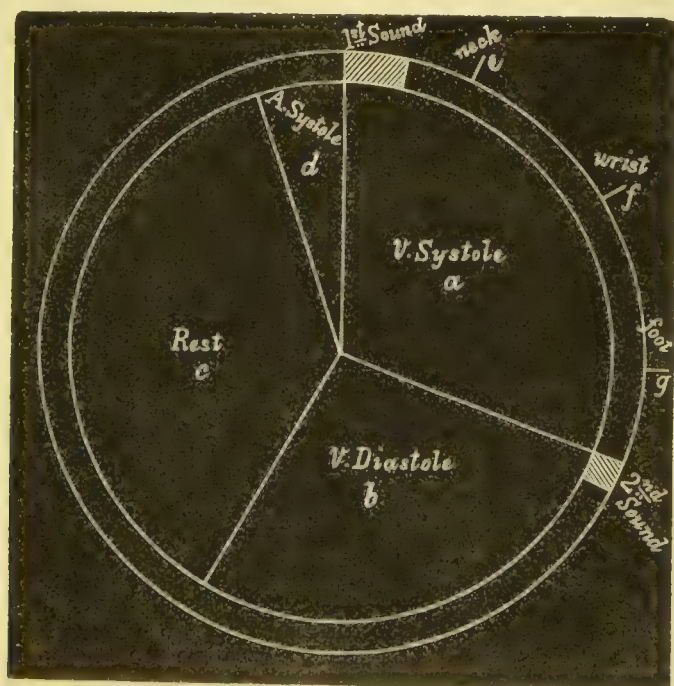
a	Period of activity	..	..	..	..	=	$\frac{1}{2}$	cycle.
b	First stage	...	...	...	...	=	$\frac{1}{10}$	cycle.
c	Second stage	...	...	...	...	=	$\frac{4}{10}$	cycle.
d	Period of repose (third stage)	...	...	...	...	=	$\frac{1}{2}$	cycle.

\* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratique*, Paris, 1868.

as the result of their experiments upon the horse, divides the cycle into two principal and equal portions (Fig. X.) ; viz., a "period of activity" and a "period of repose." The former includes two stages; the first stage comprising the contraction of the auricles, and the active diastole of the ventricles. This stage is equal to one-tenth of the cycle, or of one second, the rate of the pulse being sixty per minute. In the second stage of this period, which extends over four-tenths of a second, the pulse rate being sixty, occur, the contraction of the ventricles and the impulse, which are of equal duration ; first sound ; closure of auriculo-ventricular valves, and passage of blood into aorta and pulmonary artery.

The "period of repose," pulse being at sixty, equals five-tenths of a second, or of the whole cycle, and corresponds to

FIG. XI.



- a* Ventricular systole.
- b* Ventricular diastole.
- c* Rest.
- d* Auricular systole.
- e* Arterial pulsation in neck.
- f* " " in wrist.
- g* " " in foot.

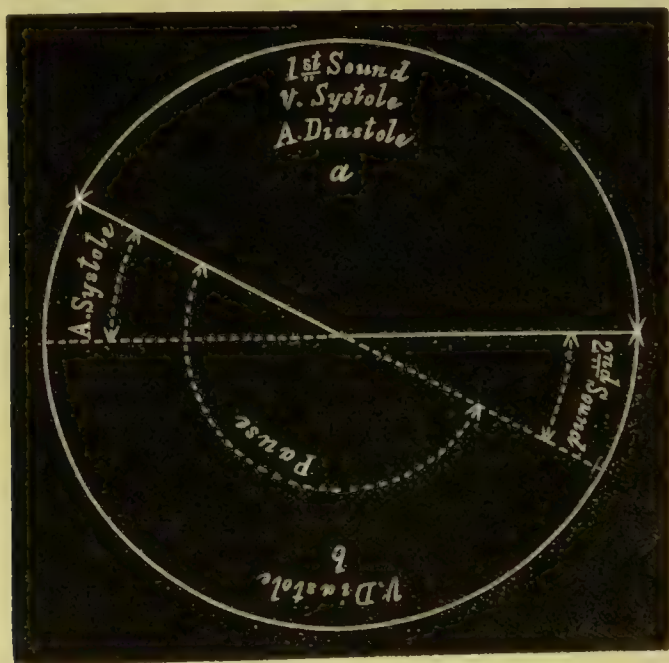


the third stage of cardiac movement. In it are included the passive diastole of the auricles and ventricles; closure of sigmoid valves, and second sound.

The great eminence of the physiologists who are the authors of this scheme entitle it to a ready and favourable reception, irrespectively of its intrinsic merits, which are very considerable. In this sketch of their opinions, however, there are some cardinal errors, which, as will be shown further on, have been since eliminated from their scheme.

Gairdner (Fig. XI.) divides the cycle into three nearly equal portions; viz., ventricular systole, ventricular diastole, and rest. The period of ventricular systole includes the first sound, cardiac impulse, and arterial pulsation in neck, wrist, and foot, in succession. Ventricular diastole embraces the second sound; whilst rest is neutral as regards sensible phenomena, but includes, at its termination, the systole of the auricles. This scheme possesses many and great merits, prominent amongst which is the recognition, for the first time made in diagrammatic form, of the true

FIG. XII.



- |   |   |     |     |   |
|---|---|-----|-----|---|
| <i>a</i> First sound, V. systole, A. diastole | * | ... | ... | * |
| <i>b</i> Ventricular diastole                 | * | ... | ... | * |

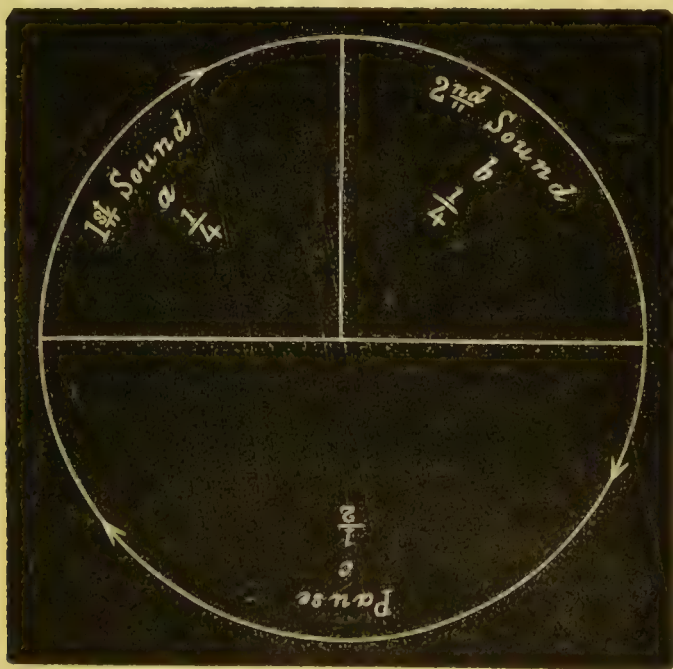


relative position of auricular systole. The apportionment of time as represented, is, however, notably incorrect.

Dr. Markham (Fig. XII.) represents the cycle as divisible into three unequal portions, viz, that of the first sound, amounting to about two-fifths of the whole, during which, and nearly synchronous with it, the impulse of the heart, the pulsation of the arteries in the neck and wrists, the systole of the ventricles, and the diastole of the auricles take place: that of the second sound, corresponding to the commencement of ventricular diastole, and the recession of the apex after the impulse, and occupying somewhat less than one-fifth of the cycle; and finally the pause, extending over somewhat more than two-fifths, or about one-half the cycle, and during which the completion of ventricular diastole and auricular systole take place.

The ventricular systole, the first sound, and the auricular diastole, although synchronous, are not coeval; that is, they com-

FIG. XIII.



*a* First sound =  $\frac{1}{4}$  cycle.

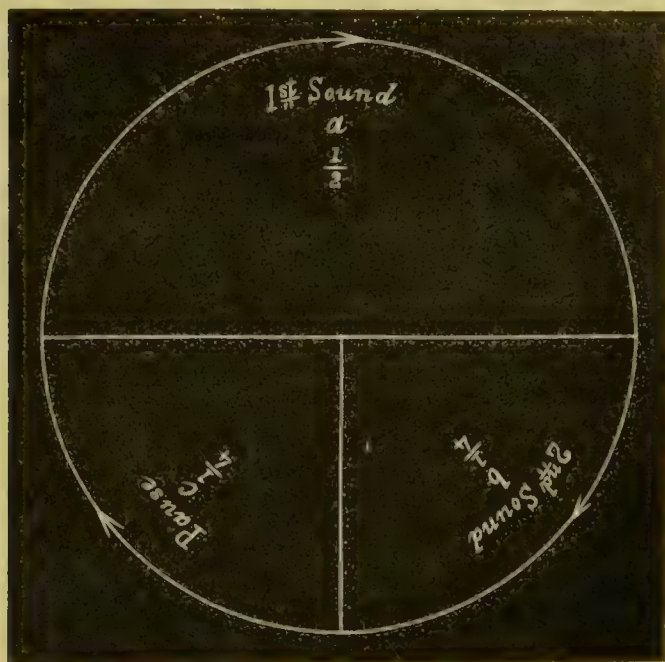
*b* Second sound =  $\frac{1}{4}$  cycle.

*c* Pause ... =  $\frac{2}{4}$  cycle.

mence but do not terminate at the same time. The systole of the ventricles and the diastole of the auricles cover the entire period, whereas the first sound occupies only the initial portion of it. The foregoing objections are applicable to this system, irrespectively of the mal-apportionment of time which may be likewise urged against it; but it has the great merit of having been the first promulgated in the English language in which an accurate appreciation of the rhythm of auricular systole is indicated.

Dr. Pavy's scheme (Fig. XIII.) consists in a division of the cycle into three parts, viz., that of the first sound, that of the second sound, and that of the pause. Each of the two former occupies one-fourth, and the latter one-half the cycle. Within the period of the first sound may be noted the impulse of the auricles; the contraction of the ventricles; tension of auriculo-ventricular valves; distension of the aorta and pulmonary artery; tilting forwards and to the right side, of the apex; and the

FIG. XIV.



- |   |              |     |     |   |                      |
|---|--------------|-----|-----|---|----------------------|
| a | First sound  | ... | ... | = | $\frac{1}{4}$ cycle. |
| b | Second sound | ... | ... | = | $\frac{1}{4}$ cycle. |
| c | Pause        | ... | ... | = | $\frac{1}{2}$ cycle. |

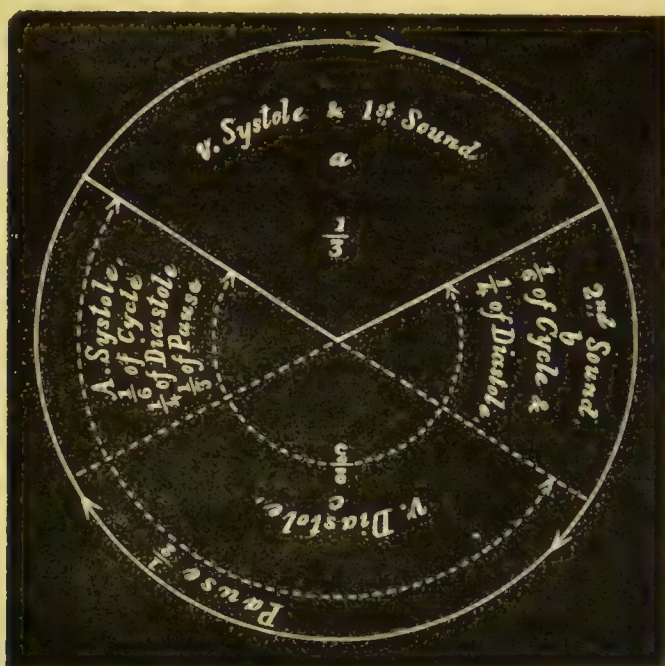
impulse of the heart. During the period of the second sound reaction of the aorta and pulmonary artery occurs; flapping down of the semilunar valves; contraction-wave of auricles towards ventricles; incipient relaxation of, and entrance of blood into, ventricles. The period of pause corresponds to the complete relaxation and gradual distension of the ventricles, and the closure of the auriculo-ventricular valves.

The relative proportion of time assigned to the sounds in this scheme is obviously incorrect, and the active contraction of the auricles at the end of the pause is not recognized.

Halford (Fig. XIV.) likewise adopts the tripartite division of the cycle, assigning, however, to the first sound one-half, and to the second sound and the pause, one-fourth respectively.

This scheme is open to the objections urged against that last described; and further, the author does not admit a short pause. According to him, the apex of the heart does not strike the wall of the chest in ventricular systole; it is pressed downwards and backwards, and from right to left, and, the impulse at the ribs is most probably given by the fibres just above the apex. He holds that the auricles continue "filling after the second sound is heard." This can only mean that blood continues to flow into them from the cavæ, coronary, and pulmonary veins, because, the auriculo-ventricular valves being open, it most probably flows as quickly from, as into, the auricles. He considers that the blow or impulse of ventricular systole is due to "pressure of the opposing column of blood on the upper surface of the semilunar valves." The pressure alluded to must be comparatively insignificant, as it is shown to be in Marey's tracings. The volume of blood sent out by the preceding contraction of the ventricles has already circulated through the pulmonary and systemic vessels; the reaction of the arteries has long since taken place, and been succeeded by the protracted period of arterial quiescence corresponding to the long pause. The pressure on the sigmoid valves by the opposing column of blood, at the moment of ventricular systole, must therefore be quite inadequate to explain, on the principle of collision of opposing forces, the systolic impulse of the ventricles. With Billing, Rouannet, Bryan, Valentin, Baumgarten, and Brakyn, he holds that the first sound is entirely

FIG. XV.



- a* Ventricular systole and first sound ... .. =  $\frac{1}{3}$  cycle.  
*b* Second sound ... .. =  $\frac{1}{6}$  cycle.  
*c* Ventricular diastole <.....> ... .. =  $\frac{2}{3}$  cycle.

valvular; and more prolonged than the second, owing to vibration of the chordæ tendinæ and valves, which are unequal in size. He thus denies to the impulse any share whatever in the causation of the first sound. In this opinion also I disagree with him.

Hyde Salter\* (FIG. XV.) adopts the natural division of the cycle into ventricular systole, and ventricular diastole. To the former he assigns one-third, and to the latter two-thirds of the whole. The first sound is represented as of equal duration with ventricular systole, which is an obvious error, as a pause or period of silence, which I designate the "systolic pause," immediately succeeds the first sound, intervening between it and the second sound, and therefore of systolic rhythm. The second sound occurs within the period of diastole, one-fourth of which,

\* The death of this eminent man has just been announced (September, 1871), and I discuss his theory under a sense of depression at the great loss medicine has thereby sustained.

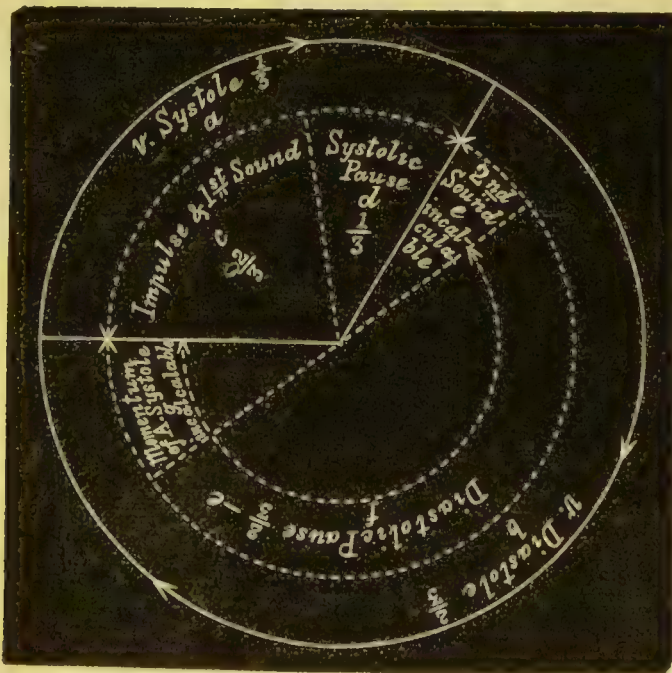


and one-sixth of the entire cycle, it represents. The pause, which coincides with ventricular diastole *minus* the second sound, and corresponds to one-half the cycle, immediately succeeds the second sound, and includes towards its termination the auricular systole, which therefore is diastolic in rhythm. This latter equals one-third of the pause, one-fourth of the diastole, and one-sixth of the cycle.

This scheme is in most respects faithful to nature, and correct in detail. I have already indicated one ground of exception to it, and in the scheme which follows others will be implied, which a comparison of the two figures (XV. and XVI.) will make sufficiently clear.

The plan which, after long and matured study, I have adopted, as most accurately representative of the natural grouping and

FIG. XVI.



a Ventricular systole	*	...	...	...	...	*	= $\frac{1}{3}$ of cycle.
b Ventricular diastole	*	...	...	...	...	*	= $\frac{2}{3}$ of cycle.
c Impulse and first sound	...	...	...	...	...	...	= $\frac{2}{3}$ ventricular systole.
d Systolic pause	...	...	...	...	...	...	= $\frac{1}{3}$ ventricular systole.
e Second sound	...	...	...	...	...	...	in calculable.
f Diastolic pause	<	.....	.....	.....	.....	>	= $\frac{2}{3}$ cycle less 2nd sound.
g Momentum of auricular systole	...	...	...	...	...	...	in calculable.

succession of phenomena, constituting a cycle of cardiac movement, is set forth in the engraving (Fig. XVI.) As therein exhibited, the cycle is divided into two primary parts, viz.: ventricular systole, occupying one-third; and ventricular diastole, occupying two-thirds of the cycle. Of the former, the first two-thirds are covered by the impulse and the first sound, and the last third by the short or systolic pause. Of the period of ventricular diastole, the initial and the terminal portions are occupied, respectively, by the second sound, and the momentum or most energetic portion of auricular systole, both of which are incalculable as to duration. Inasmuch as, in the normal state, the contraction of the auricles is not manifested by any sensible phenomenon, it is conveniently included within the period of the long or diastolic pause, which commences at the end of the second sound, and is characterized by the absence of all tactile and acoustic signs.

Within the period of the first two-thirds of ventricular systole, coinciding with the impulse and the first sound, the arterial valves are thrown open; the auriculo-ventricular valves are shut down; the aorta and the pulmonary artery are distended by efflux from the ventricles; and the auricles, partly by arrest at the auriculo-ventricular openings, and partly by influx from the cavæ and pulmonary veins, are likewise engorged. The circulation in the coronary arteries is retarded; and that of the coronary veins is accelerated. The pulsation of the great arteries of the neck is virtually coincident with the impulse and the first sound, although necessarily, by a brief but inappreciable interval, posterior to them in time.

During the short pause which immediately succeeds the first sound, and precedes the second sound, and which, according to Marey, equals in duration one-tenth of a second, the contraction of the ventricles and the distension of the auricles and arteries continue; the arterial valves are still open, and those of the auriculo-ventricular orifices still firmly closed; temporary stasis of the circulation in the cavæ and their immediate tributaries, and in the pulmonary veins, takes place; the circulation in the coronary arteries is still retarded, whilst that in the coronary veins is quickened, and the radial pulse is perceptible. In

slow action of the heart the short pause may be readily recognized; in rapid action, and even at the ordinary rate, it is not so easily appreciated, and hence it has been, by many excellent observers, entirely ignored. Murmurs are occasionally, and not unfrequently, heard, which are most appropriately designated "post-systolic" and "pre-diastolic," as occupying, the former the initial, and the latter the terminal portion of this pause or period of silence. To this subject I shall have occasion to return. Dr. Sibson regards the short pause as coinciding with the commencement of ventricular diastole, and the reaction of the arteries; he says\*: "A little consideration will make it evident that the sigmoid valves must come together at the very end of the systole, otherwise blood would at once flow back into the ventricle, during a period that, as M. Marey has shown in his important work on the circulation of the blood, precedes the second sound by one-tenth of a second, and coincides with the short pause. During this short pause between the first and second sounds, the walls of the arteries, just charged to the full by the systole, return upon themselves and drive the blood forwards into the smaller vessels, and backwards upon the arch and aortic valves." I believe, as already stated, that the ventricles, during the short pause, are still in a state of contraction, and the aorta and pulmonary artery in a state of distension by the ebbing current of blood; hence reflux cannot take place, though the valves be still open. MM. Chaveau and Favre conclude from their experiments, that ventricular diastole commences *with* the second sound, whereas the short pause is antecedent to it.† Coincident with the second sound is reaction of the aorta and pulmonary artery, closure of the arterial, and opening of the auriculo-ventricular valves, relaxation of the ventricles, and passage of blood from the auricles into the ventricles. The period occupied by the second sound is incalculably short. The long or diastolic pause succeeds the second sound, and with it covers two-thirds of the cycle. During this pause the arterial valves are still closed, and the auriculo-ventricular valves open; the ventricles

\* *The Movements, Structure, and Sounds of the Heart*, 1869, p. 31.

† *Traité de Physiologie*, par F. A. Longet, 3ème Edition, tome, 2ème, 1869, p. 156.



are relaxed and in process of dilatation ; undulatory contraction of the auricles, from the appendices towards the auriculo-ventricular openings, sets in and is continued, with passage of blood into the ventricles ; and at its conclusion the auricles perform a brief but energetic act of contraction, the *momentum* of auricular systole, by which the ventricles are filled to distension, and reaction of their walls, constituting the succeeding ventricular systole, is induced.

I may here briefly state the result of my observations on M. Groux, who exhibited in his own person the pulsations of the heart, through a congenital fissure of the sternum, in Dublin, in the year 1858. On the 29th of February in that year I made the following notes. There was a vertical sulcus in the situation of the sternum, extending nearly its entire length, and bounded on each side by a continuous ridge of bone representing the margins of the sternum. The ensiform cartilage, or a flexible substance representing it, occupied the lower extremity of the fissure, which, at its widest part about midway between the upper and lower extremity, was an inch and a-half in transverse diameter ; it was, however, susceptible of expansion to three inches by retraction of the shoulders, and by forced inspiration. Within this fissure a pulsating tumor existed, which was irregularly ovoid in figure, one and a half inch in its long, and half an inch in its short diameter, and did not extend laterally beneath the boundaries of the fissure. The long axis of this tumor extended from below and the left side upwards and to the right. The upper portion of the fissure was occupied by another and smaller pulsating tumor, seemingly continuous with the former. The superior extremity of the former of these tumors was on a level with the upper edge of the third costal cartilage, and its lower extremity with the middle line of the third intercostal space. During the period of enlargement this tumor communicated to the hand a feeling of increased hardness, and a rolling motion from above and right side downwards and to left ; its pulsation was synchronous with the first sound, and followed by the second sound. The superior of these tumors pulsated first, and in an arc from above downwards and to the right, and the lower pulsation preceded the carotid and radial pulse.



Judging from the extent and course of the pulsation-wave, and from what may be observed in the dog, I regarded the inferior pulsating tumor as belonging to the right ventricle. The right auricle touches the anterior wall only by its appendix, and with a sudden "tap," during its diastole; it was soft at the moment of greatest distention; whereas, the line in which the remainder of the auricle presents itself to the anterior wall of the chest is from above downwards and to the right side. The superior tumor was the right auricle, which contracted first, in order of time, after the long pause, conducting a wave of blood into the right ventricle, the contraction of which immediately succeeded that of the auricle.

Dr. Pavy\* holds that an ascending wave travels over the auricle immediately after, and communicated by, the systole of the ventricle; that this is promptly succeeded by a descending auricular wave running into the ventricle; that then the pause of ventricular diastole occurs, and next ventricular systole.

Dr. Pavy is, therefore, of opinion that in mammals, including man, auricular systole immediately *succeeds* instead of immediately *preceding* the systole of the ventricles, and that in this class no other contractile movement of the auricles occurs, although such is not the case in the frog and the tortoise.

I take the liberty of declaring that herein Dr. Pavy, excellent observer though he be, has fallen into error. An ascending wave of vibration, communicated to the blood of the auricle by the shock of ventricular systole through the tense auriculo-ventricular valves, is, no doubt, exhibited; it is equally true that a descending wave of contraction immediately succeeds the former, and conducts the first gush of blood into the ventricle after the retraction or opening of the auriculo-ventricular valve, or rather by the pressure of which mainly it is thrown open. The long pause now ensues; during its continuance blood still flows passively into the auricle and ventricle respectively; but now, towards the close of the long pause, auricular contraction of a more abrupt and vigorous character takes place, filling the ventricle to distention, and provoking an act of ventricular systole in which it is itself merged, and so on. This *presystole* or auri-

\* *Medical Times and Gazette*, 21st November, 1857.

cular systole immediately preceding ventricular systole, is of the utmost moment diagnostically, as will appear in the sequel. I shall place in parallel columns, for the purpose of comparison, the succession of events as represented by Dr. Pavy and by myself.

## PAVY.

Ventricular systole.  
 Ascending auricular wave (vibratory).  
 Descending auricular wave (contractile).  
 Pause of ventricular diastole.

## HAYDEN.

Ventricular systole.  
 Ascending auricular wave (vibratory).  
 Descending auricular wave (contractile).  
 Pause of ventricular diastole (long pause).  
 Auricular systole (momentum of).

Between Dr. Pavy's views and mine, then, the difference consists in the denial by him of a continuous descending wave of auricular contraction, during the entire period of ventricular diastole; and, what is still more important, his repudiation of pre-systolic auricular contraction, both of which events I strongly insist upon.

The causes to which the two sounds of the heart have been respectively attributed by different authors, will be found briefly set forth in the subjoined table taken from *Longet's Physiology*,\* and borrowed by that author from Barth and Roger. This table I have supplemented by the addition of the views of Dr. C. J. B. Williams, Dr. Bellingham, M. Chauveau, and my own.

*First sound.**Second sound.*

Laennec, ...	Contraction of ventricles.		Contraction of auricles.
Turner, ...	Contraction of ventricles.		Shock of the heart rebounding against the pericardium in diastole.
Corrigan, ...	Shock of the blood against the walls of the ventricles in diastole.		Collision of the internal surface of the walls of the ventricles in systole.
D'Espine, ...	Contraction of the ventricles.		Dilatation of the ventricles.
Pigeaux(1832),	Shock of blood against walls of ventricles at the moment of diastole.		Shock of blood against the walls of the aorta and pulmonary artery at the moment of systole.
Pigeaux(1839),	Friction of the blood against the walls of the ventricles, the orifices and walls of the great vessels in systole.		Friction of the blood against the walls of the auricles, the auriculo-ventricular orifices, and the cavity of the ventricles, in diastole.
Hope (1834),	Mutual collision of the corpuscles of the blood in systole.		Mutual collision of the corpuscles of the blood, and impulse of blood against the walls of the ventricles, in diastole.

\* Vol. ii., p. 153.

	<i>First sound.</i>	<i>Second sound.</i>
Hope (1839),	The sound of valvular tension, and of muscular extension, in systole.	The click of the sigmoid valves in diastole.
Rouannet, ...	The click of the auriculo-ventricular valves in systole.	The click of the sigmoid valves in diastole.
Piorry, ...	The entrance of blood into the left chambers of the heart.	The entrance of blood into the right chambers of the heart.
Piédagnel, ...	The contraction of the left ventricle.	The contraction of the right ventricle.
Carlile, ...	The sudden entrance of blood into the great arteries in systole.	The click of the sigmoid valves in diastole.
Magendie, ...	The shock of the apex of the heart against the side in systole.	The shock of the anterior surface of the heart in diastole.
Burdach, ...	Sudden entrance of blood into the ventricles containing air, in auricular systole.	The injection of blood into the arteries containing air, in ventricular systole.
Bouillaud, ...	The sudden tension and mutual collision of the auriculo-ventricular valves, and the sudden displacement of the sigmoid valves, in systole.	The sudden tension and shock of mutual collision of the sigmoid valves, and the sudden raising of the auriculo-ventricular valves, in diastole.
Gendrin, ...	Vibration resulting from the collision of the blood in systole.	Percussion of the blood against the walls of the ventricles in diastole.
Cruveilhier, ...	The sudden raising of the sigmoid valves in systole.	The lowering of the sigmoid valves in diastole.
Skoda, ...	The shock of the blood against the auriculo-ventricular valves, and the impulse of the apex against the side. The shock of the blood against the walls of the aorta and pulmonary artery, in systole.	The shock of the column of blood against the walls of the ventricles, and the retrograde shock of the column of blood against the sigmoid valves, in diastole.
Beau, ...	The shock of the wave of blood against the walls of the ventricles, in ventricular diastole.	The shock of the column of blood arriving from the veins against the walls of the auricles.
G. Williams,	Muscular contraction of the ventricles in systole.	The shock of the returning column of blood against the sigmoid valves, in diastole.
Dublin Committee, 1835,	Friction of blood against the walls of the ventricles, and muscular contraction, in systole.	Tension of the sigmoid valves, and shock of the returning columns of blood, in diastole.
London Committee, 1836,	Sudden muscular tension of the ventricles, and shock against the chest, in systole.	Sudden closure of the sigmoid valves by the columns of blood in the arteries (in diastole).
Philadelphia Committee,	Muscular contraction of the ventricles, and click of the auriculo-ventricular valves in systole.	Closure of the sigmoid valves by the shock of the returning columns of blood in the aorta and pulmonary artery.

*First sound.**Second sound.*

C. J. B. Williams,	Muscular contraction of ventricles, closure of auriculo-ventricular valves, and occasionally impulse of heart.	Click of sigmoid valves.
Bellingham,	Rush of blood through arterial orifices.	Rush of blood through auriculo-ventricular orifices.
Chauveau, ...	Sudden tension of auriculo-ventricular valves.	Sudden tension of sigmoid valves.
Hayden, ...	Shock of apex against wall of chest, and sudden tension of auriculo-ventricular valves and chordæ tendineæ.	Sudden tension of sigmoid valves.

M. A. Dumontpallier gives the following summary of the experiments of Chauveau, performed at Alfort on the 5th of September, 1860, in his presence, and repeated on the 29th of the same month in the presence, amongst others, of Doctors Corrigan and Banks of Dublin, with a view to determine the nature and cause of the sounds of the heart.\*

A horse having been pithed, and artificial respiration maintained by means of a pair of bellows connected with the trachea, a "window" was made in the left side of the chest, and the pericardium laid open through this, so as to expose to view the left ventricle, left auricle, and the pulmonary artery. The auricle was seen to contract very feebly; but the ventricle with much energy, contracting from apex to base, and becoming globular, hard, and corrugated on the surface. The left wall of the ventricle was forcibly elevated in such a manner that the organ must have struck the wall of the chest. A stethoscope applied over the ventricle enabled the observer to hear two sounds; the first dull and prolonged, and coinciding with ventricular systole; the second sharper and shorter, coinciding with ventricular diastole.

After the first sound "a short silence" ensued, and after the second, "a longer silence." When the root of the pulmonary artery was taken gently between the finger and thumb at the moment of the second sound, a vibration was felt, and a stethoscope applied here at the same moment enabled the observer to hear the second sound. If, at the moment of systole of the ventricle and occurrence of the first sound, the roof of the left auricle

\* *Gazette des Hospitaux; Dublin Hospital Gazette*, 1st November, 1860.



were pushed down with the finger, the vibration of the auriculo-ventricular valve was felt. If now an incision were made in the auricular appendix, and the finger introduced through it into the auriculo-ventricular orifice, it was felt to be grasped at the moment of systole by the free edges of the valves, and held for a period corresponding in duration to the first sound, which was either suppressed or weakened, owing to suspension or impairment of the normal vibration of the valves. The reporter adds: "From this experiment may we not conclude that the first sound is owing, almost entirely, to the contraction of the auriculo-ventricular valves?" A query which, I apprehend, few if any will answer in the negative.

Chauveau next proved, by mutilation of the valves, that abnormal sounds depend upon disease of these. Thus, when closure of the mitral valves was prevented during ventricular systole, by means of a small steel rod, blood rushed back into the auricles, and a *bruit de soufflet* was the result. Tearing of the valves, or dividing the chordæ tendineæ was followed by a similar result. Systolic murmur likewise followed compression of the pulmonary artery during ventricular systole.

In a similar manner he caused diastolic murmur by keeping the sigmoid valves apart by means of a fine instrument, by pinning them up to the wall of the artery, or by destroying them. In every such instance blood rushed back into the ventricle and produced diastolic murmur.\*

Marey and Chauveau† have determined, by experiments on the horse, the relative duration and precise relationship of the several movements of the heart.

The systole of the auricles precedes that of the ventricles by two-tenths of a second. Ventricular systole and impulse commence together, but the former exceeds the latter in duration by one-tenth of a second, the length of ventricular systole being three-tenths of a second.

They have also proved experimentally that the force of contraction of the right ventricle is to that of the left ventricle, in the horse, as 1 to 3. The relative proportion of the various car-

\* See *Gazette Médicale*, 1856, for Chauveau's memoir on this subject, *in extenso*.

† *Longet's Physiology*, 1869, vol. ii. p. 146.

diac movements is, according to these eminent authorities, the same in man as in the horse.

It will be seen by reference to Fig. XVI. that the conclusions at which I had arrived independently, from observation of the healthy human heart, are nearly identical with those just detailed. I did not see the report of Marey and Chauveau's experiments till very recently (1868), and I am therefore entitled to regard them as eminently confirmatory of my own views.

## CHAPTER II.

### PHENOMENA OF THE HEART'S ACTION IN RELATION TO THEIR MODIFICATION BY DISEASE.

I SHALL now briefly discuss, individually, the cyclical series of phenomena which constitute a single action of the heart.

The impulse of the heart, which, in the state of health, may be perceived by the application of the hand in the fifth intercostal space of the left side, about two inches below the level of the nipple, and one inch inside the nipple-line, is characterized by a gentle lifting motion over an irregularly circular space of about one inch in diameter. This is due to the impact of the apex and adjacent portion of the anterior surface of the left ventricle, against the chest wall; commences with the systole of the ventricles, which exceeds it in duration by one-third; and strictly corresponds in time with the first sound. As the systole is equal to one-third of the cycle, so the impulse equals one-third of the diastole.

There are at least three positive varieties of abnormal impulse, besides those of a negative character, namely: the prolonged, diffused, heaving, and *double* impulse of left ventricular *dilated* hypertrophy; the abrupt but prolonged and heaving impulse of left ventricular *simple* hypertrophy; and the abrupt hammering impulse of nervous palpitation.

The impulse is liable to impairment, suppression, and displacement. It is obscured or enfeebled in fatty degeneration of the heart, and in atrophic attenuation of the left ventricular walls. In the advanced stages of the former of these conditions it may be entirely suppressed, and likewise in the worst form of typhus fever characterized by suspension of the first sound.

*Displacement* of the apex of the heart, to whatever cause due, will involve change of the seat of impulse, and may take place in the direction upwards, downwards, towards the right, or towards the left side.

Displacement of the heart to either side is most frequently the consequence of liquid or gaseous effusion into the opposite pleura. Two different opinions are held as to the mode in which this displacement is effected. One, and that which is almost universally accepted, is that the heart is displaced by mechanical *detrusion* to the opposite side; and the other, to the effect that it is *drawn* towards the sound side by the elastic tension of the unaffected lung.

This latter view has been ably advocated by Dr. Douglas Powell,\* who considers the median position of the mediastinum, in the state of health, to be maintained by a well-balanced tractive force exercised upon its opposite sides by the elastic tension of the lungs. When either lung, therefore, passes into a state of collapse, whether from liquid effusion or aeriform accumulation, the normal balance of pulmonary tension is of necessity deranged, and the mediastinum and heart are *drawn* towards the unaffected side by the unopposed tension of the sound lung. In pneumothorax the displacement is instantaneous; but he admits that "beyond this it may be increased by the direct pressure or subsequent accumulation of air." In support of this doctrine the following experiments are adduced:

*Experiment 1.* In a fresh subject, a needle was thrust through the pericardium at the left margin of the sternum; an opening was then made in the left pleura, through which air was sucked in, and immediately the needle was deflected to the left. The arms were now drawn up equally, and an act of inspiration thus artificially made after the manner of Sylvester, and the deflection of the needle to the left was observed to be immediately increased.

*Experiment 2.* A dog was brought under the influence of chloroform, a needle was inserted into the pericardium, and the pleura laid open as in the former experiment, and with a similar result, namely, deflection of the needle towards the injured side, to the extent of 20°.

It seems to me that in Experiment No. 1 the deflection of the needle to the left was directly and exclusively due to the pres-

\* *Medical Times and Gazette*, 30th January; 13th and 20th February; and *British Medical Journal*, 17th July, 1869.



sure upon the mediastinum of the air just admitted into the pleura; and the increased deflection exhibited on drawing the arms upwards was the result of increased pressure by the introduction into the pleura of a further volume of air. This air was free, and its pressure therefore not neutralized by the elastic reaction of the lung, as was the case with that included within the pulmonary tissue of the opposite lung. The right lung was, moreover, in the first experiment, in the state of maximum expiratory contraction characteristic of the dead body, and its elastic tension proportionately reduced. In this condition, the elastic reaction of the lungs of the dog was found by Hyde Salter equal to a pressure of only four inches of water.\* The elastic tension of the lung is an expiratory force, increasing with its expansion, and tending towards a common centre. No doubt this centre is in the root of the lung, but external to the mediastinum. Here, therefore, the elastic reaction of the lung has its point of rest, and beyond this in the direction inwards the range of its elastic tension does not extend. The length and extensibility of the blood vessels and air-tubes of the lungs, and their capacity for physical reaction, are strictly adapted in the normal state to the dimensions of the pleural cavity and the position of the mediastinum.

In reference to Experiment No. 2, which is in no degree less open to all the objections above-stated, I have in addition to remark, that no mention is made of oscillation of the needle from right to left alternately with inspiration and expiration, which must have taken place if deflection depended upon the movements of the right lung. As to the alleged effect of sudden introduction of air into either pleura, in causing "partial collapse of the opposite lung, by removing resistance to its elastic tension," I cannot perceive how the position of the mediastinum can be in any degree thereby influenced, save in so far as the pressure of free air on the diseased side must determine its displacement towards the opposite. Surely, even though partial collapse *per se* could in any degree influence the position of the mediastinum, complete collapse on the opposite side would be more than competent to neutralize it.

\* *Lancet*, 5th August, 1865. -

Dr. Powell published two cases\* of pyo-pneumothorax, illustrative of displacement of the mediastinum by "elastic recoil" of the opposite lung. By means of a water-pressure guage he showed that no air-pressure was exercised upon the mediastinum on the diseased side of the chest. But pus was found on that side in both cases, and in one of these to the amount of a pint.

With a view to satisfying my mind as to the validity of the objections above urged against Dr. Powell's doctrine, and as to the degree, if any, in which inspiratory expansion of either lung, the other being functionally in abeyance, was capable of influencing the position of the mediastinum, I performed, on the 14th October, 1871, the following experiment, with the assistance of Mr. Ward, one of our demonstrators of anatomy, and in presence of the anatomical class in the School of Medicine, Cecilia-street.

The anterior wall of the chest of a subject brought in for dissection was removed, with the exception of the sternum, which was divided with the saw by a median incision from the upper to the lower extremity. The edges of this incision were divaricated by means of a wedge, to the extent of an inch, and a piece of copper wire, nine inches long, made sharp at one extremity, and rolled into a coil at the opposite, was inserted perpendicularly into the pericardium, and fixed in the substance of the heart. The left lung was now detached from its root, and the mouth of the left bronchus plugged with a cork, which was held in position and made air-tight by means of a ligature passed round it. Through the outer extremity of the cork a string was passed, by means of which it might be instantaneously removed. Through an incision made in the trachea at the root of the neck a large glass tube was introduced, and the right lung slowly inflated, the subject lying upon its back. The position of the wire fixed in the pericardium was not in the smallest degree influenced till towards the point of maximum inflation, when the anterior edge of the lung advanced and began to press against the side of the pericardium. The free extremity of the wire now began to deviate to the right, and at the end of inflation had moved fully an inch towards the right side; and on allowing

\* *Medical Times and Gazette*, 21st August, 1869.

the lung to collapse by its elastic reaction, through escape of the air from the opening in the trachea, the needle gradually returned to its original position. The process was repeated several times, with the same result. At the moment of maximum inflation the cork was now suddenly withdrawn from the bronchus, and the lung thus permitted to collapse abruptly, as if by rupture of the opposite lung; but the result was in no degree altered, save in regard to the rapidity of return movement of the lever from right to left.

The obvious conclusions to be drawn from this experiment are the four following: First, that in the first stage of inspiratory expansion of the lung, the position of the mediastinum is in no degree affected; second, that towards the end of the second and final stage of expansion of the lung, the mediastinum is displaced towards the *opposite* side by pressure of the anterior edge and internal surface of the inflated lung; third, that at no stage of inspiration is the mediastinum displaced by *traction towards* the expanding lung; and, fourth, that the sudden exhaustion of one lung, even in the stage of maximum expansion by rupture of the opposite lung, is incapable of determining by recoil or otherwise, a movement of the mediastinum towards that side, beyond the point of equilibrium.

Having now shown, as I conceive, good reason for adhering to the doctrine of lateral displacement of the heart by pressure, as promulgated by Bertin, Laennec, Hope, Stokes, Walshe, and indeed by the great majority of writers, and as almost universally held at the present time, I proceed, after the example of Stokes,\* to classify all cardiac displacements whatsoever under the two heads of "excentric" and "concentric."

The former are caused by diseases "of accumulation," solid, liquid, or gaseous, external to the pericardium, and pressing *ab extra* upon the heart.

The latter are due to contraction in volume of either lung, and a concentric movement of adjacent parts, including the heart, to fill the vacuum thence potentially arising.

The excentric displacements, as the name implies, are therefore dependent upon a force operating in a direction from its

\* *Opus citat.*, p. 458.



seat; whilst those named concentric are no less obviously due to a cause acting by traction towards the same point. Excentric displacements may take place in the four opposite directions, upwards, downwards, to the right, or to the left side; and diagonally or intermediately to these lines. Upward displacement, direct or oblique, of the heart by pressure, is most frequently caused by enlargement of the left lobe of the liver, hydatid tumor of that organ, perihepatic abscess, aneurism of the inferior thoracic aorta,\* and temporary paralysis of the left ala of the diaphragm, with ascent of an inflated stomach in diaphragmatic pleuritis.†

Bouillaud declares that upward displacement of the heart may be caused by ascites in an extreme degree.‡ I have never witnessed notable elevation of the heart from this cause. Displacement downwards is, when present, usually the result of pulmonary emphysema engaging pretty equally the anterior portion of both lungs; by the diagonal pressure so exercised, as well as by the general depression or flattening of the diaphragm, the heart is displaced downwards, and occupies a position behind the xiphoid cartilage. Downward displacement may likewise result from the pressure of an aneurismal or other tumor occupying the upper portion of the anterior mediastinum.

Excentric displacement to either side is most frequently due to liquid accumulation in the opposite pleura. In the effusion consecutive to acute inflammation, Dr. Stokes has shown, that the relaxation of the mediastinum involved in the acute inflammatory process may determine lateral displacement of the heart at an early period of the disease, and under the comparatively slight pressure of an undistended pleura. In all such cases the heart is dislocated in the horizontal line, and occupies a position under the right nipple when the effusion is on the left side; and when on the right, the heart may be felt pulsating at a greater or less distance outside the left nipple-line. Doctor Douglas Powell has shown§ that in the right lateral displacements the

\* See case (Fay), chapter ix.

† For a case in illustration, see an article by the author, entitled "Diaphragmatic Pleuritis," *Dublin Medical Journal*, August, 1871.

‡ *Traité des Maladies du Cœur*, tom. ii., p. 531.

§ *British Medical Journal*, July 17, 1869.



apex of the heart is depressed, but never so as to occupy a position external to the base ; whereas, in displacements to the left side, the apex is relatively elevated, and the long axis of the heart nearly or quite horizontal.

Cases of this kind are characterized by percussion dullness on the side whence the heart has been displaced, or posterior in the line of transit of the organ, and commensurate with the extent of its displacement ; by clearness on the opposite side in advance of the organ ; and where the liquid is absorbed or otherwise removed, by return of the heart to its natural position with corresponding rapidity.

The functions of the heart and the attendant phenomena are in no degree modified by its abnormal position, with the following exceptions : when placed in front of, and in contact with a solid tumor, such as an aneurism, a solidified lung,\* or the spinal column,† its impulse may be so vigorous and diffused as to lead to the diagnosis of hypertrophy. Hope has also noted, in a case of dextrocardia, the existence of systolic murmur, which ceased on the return of the heart to its normal position ; and was due, as he believed, to twisting of the aorta in its altered relationship to the heart.‡

Consecutively to the removal of the displacing fluid by absorption or otherwise, the heart not unfrequently has been known to transgress its normal boundary, and pass considerably to the opposite side. This occurs where the lung has been compressed, and rendered incapable of expansion, after removal of the compressing medium, by previous obliteration of its vesicular structure ; or by the constricting agency of dense false membrane. Such a phenomenon is, *mutatis mutandis*, analogous to the retraction of the features from consecutive atrophy and shortening of the paralysed muscles, in unilateral paralysis of the face. The distinctive features, then, of lateral displacement of the heart by liquid accumulation in the opposite pleura, are, the slow and mensurable mode of its occurrence ; dullness on the side whence it has been displaced, and clearness on the opposite side beyond the limits of cardiac dullness ; and in the event of removal of the displacing medium, return of the heart to its natural situation ;

\* Stokes.

† Hope.

‡ *Opus citat.*, p. 536.

or beyond it where the lung, previously compressed, has become incapable of expansion.

Lateral displacement of the heart, by detrusion, may be likewise caused by cancerous growths in connection with either lung or pleura. In Dr. Houston's case a cancerous tumour of the right lung was the sole cause of displacement of the heart to the left side.\*

In most cases, however, of displacement of the heart by pressure connected with cancerous growths of the lung or pleura, but especially the latter, serum was likewise present in large quantity, and presumably was the principal agent in displacement. Case 20 (Rourke), affords a notable example of this kind. Where, however, the tumour is of the nature of soft or medullary cancer, which, by its expansion and elasticity is capable of exercising diastolic pressure, displacement of the heart may be, and usually is, effected without the intervention of liquid accumulation. Cancerous disease of the lung or pleura, operating as a cause of displacement of the heart, and associated or not with serous effusion, is characterized by certain symptoms of great diagnostic value; these are especially the two following, namely, engorgement of the subcutaneous veins of the affected side of the chest; and distinct transmission, over the same side, of the sounds of the heart. Associated with dullness on percussion; absence of respiratory sound save at the root of the lung, and of vocal fremitus; and with excentric displacement of the heart; the signs above indicated become pathognomonic of cancer of the lung or pleura.

Thoracic aneurism may be likewise a cause of displacement of the heart to either side, but most frequently the right, because when capable of making lateral pressure upon the heart, its usual situation is the posterior mediastinum and left pleural cavity. The diagnosis of this cause of displacement of the heart cannot be difficult.

The heart may be likewise displaced to either side by the pressure of free air in the opposite pleural cavity, constituting pneumothorax; by pulmonary emphysema preponderating on one side, and engaging the anterior and inner portion of the lung; and by passage of the hollow abdominal viscera into the thorax.

\* See *Catalogue of Museum of Royal College of Surgeons, Ireland.*

Dr. Stokes is of opinion that simple pneumothorax, without fistula in the lung, can cause displacement of the heart only when the aeriform collection is large in quantity; and that, with fistula, it can produce this result only where the egress of air is prevented during expiration, that is, where the fistula is valvular.

I incline to regard this view as somewhat too exclusive. Free air in the cavity of the pleura, with or without fistula of the lung, and at the temperature of the living body, must undergo considerable expansion during inspiration, and a high degree of compression during expiration. In the former condition the mediastinum, as the most yielding wall of the hemithorax, is liable to undergo displacement under the pressure of elastic expansion of the air, no longer neutralized by the resiliency of the lung and pulmonary pleura; and in the latter state, or that of expiration, under the compressing force of the reacting thoracic walls the contained air must undergo a high degree of condensation, and the mediastinum must sustain an equivalent pressure on the corresponding side. These forces, in succession, would operate continuously. A fistular opening in the lung of the average diameter, even though direct and patulous, is by much too narrow to neutralise, though it may modify this pressure, by evacuating the pleura within the period of an ordinary expiration. In order to effect this, it must bear the same proportion to the pleural cavity which the trachea does to the expanded lung; and even in that case a certain degree of obstruction to the exit of air would be necessarily presented at the fistula, and an equivalent centrifugal pressure sustained by the parietes of the pleural cavity, owing to the absence of the gradual and well-adapted transition of cubic space exhibited by the bronchial system in its relationship to the trachea.

Simple pneumothorax, then, is of itself, and *a fortiori* in conjunction with liquid, in my opinion competent to effect lateral displacement of the heart. After death, owing to the cooling and condensation of the air within the pleura, the heart will have returned in some degree towards its natural situation. In such a case, therefore, the position of the heart, as exhibited by *post mortem* examination, will not accurately represent its situation at the time of death. At the latter period the dis-

placement is greater, owing to the alternate expansion and condensation of the æriform contents of the pleura, under the combined influence of heat and compression. Emphysema of the lungs, if greatly in excess on one side, and engaging the anterior portion of the organ, may become the agent in lateral dislocation of the heart; but this affection is usually bilateral, and pretty equally balanced on both sides of the chest; hence the cardiac displacement resulting therefrom consists usually in depression of the organ as previously mentioned. Increase in the volume of the lung, and consequent displacement of the heart, may be excluded from the category of results of inflammation of the substance of the organ. Nevertheless, an example has been adduced by Professor R. Smith\* of such increase in the volume of the right lung in "plastic pneumonia" that the liver and diaphragm were considerably depressed into the abdomen. In such a case the heart could hardly escape displacement. Diaphragmatic hernia, involving intrusion of the abdominal viscera into the chest, completes the category of causes of lateral displacement of the heart. Comparatively few examples of this singular malposition are on record, and these have been all associated with partial deficiency of the diaphragm.

Bouillaud quotes from Weyland the following illustrative cases:

"In an infant which died some hours after birth, cadaveric examination revealed that the stomach, a great part of the intestines, the spleen, and the pancreas, were contained in the left side of the chest. The right side included the heart, the thymus, and the two lungs."

"An infant of seven years died of vomiting, which had continued from its birth, and ultimately became complicated with cerebral disease. On opening the body it was found that the cavity of the left side of the chest, to the level of the second rib, was filled with the convolutions of the intestines. The heart was situated in the middle line. The left lung, atrophied, presented scarcely a sixth part of its normal volume. The right lung was well formed, and presented nothing unusual."†

The remarkable case of phrenic hernia with displacement of

\* *Dublin Journal of Medical Science*, vol. xix.

† *Traité Clinique des Maladies du Cœur*, 1835, tom. ii. p. 524.



the heart to the right side, from congenital imperfection of the diaphragm in a female aged 75, recorded by Cruveilhier,\* deserves special notice, as being in many respects the most singular example of the kind to be found in medical literature.

The following remarkable example of congenital malposition of the abdominal and thoracic viscera occurred in the practice of Dr. Osborne, and has been quoted by Drs. Stokes and Graves.† I give it in the words of Dr. Stokes :

“A man about 40 years of age died of tubercular phthisis (in Sir Patrick Dunn’s Hospital). The œsophagus, after passing through the usual opening in the diaphragm, was found to re-enter the thorax by another very large opening in the tendinous expansion towards the left side. The stomach, of which the cardiac and pyloric extremities were approximated, occupied the inferior portion of the left thoracic cavity.

“A considerable portion of the transverse arch of the colon was also included in the left side of the chest. These viscera, loosely but permanently fixed by means of the serous membranes, all rested on the convex surface of the diaphragm, and had pushed the heart and mediastinum towards the right side.

“The margin of the unnatural opening in the diaphragm was formed by a round tendinous cord, about the thickness of a quill, which added greatly to its strength, and was evidently of very ancient formation. The lung, small and tuberculated, did not exhibit any signs of compression, and was adherent to the abdominal viscera. It may be easily conceived that the left pleural cavity was continuous with the cavity of the peritoneum, and both were lined by the one serous membrane.”

Dr. Stokes remarks, “This case is pregnant with interest; we observe in the adult *a new cause of displacement of the heart*, and a new source of difficulty in stethoscopic examination; for it is quite evident that auscultation applied to the left side of the thorax would have furnished very fallacious information, and the sounds heard would have varied according as the stomach and colon were full or empty. The same observation applies also to percussion; and the fact is that during the life of this

\* *Anatomie Pathologique*, livraison 17.

† *Dublin Hospital Reports*, vol. v.

patient, those who examined his chest could not reconcile the phenomena afforded by auscultation or percussion with any known disease of the chest. The respiration was heard everywhere, except inferiorly and anteriorly on the left side, and here percussion gave a clearer sound than natural. No râle was audible in this part of the chest, but borborygmi and sounds resembling those produced by the motion of fluids in the intestines were observed.”\* This is the case on which Graves and Stokes with good reason dwell as affording conclusive evidence of the competency of the stomach to accomplish the act of vomiting, unaided by the abdominal muscles, as vomiting had repeatedly occurred during the man’s last illness. Dr. Stokes makes the important remark, “ In the case of imperfectly developed diaphragm, in congenital dextrocardia, and again, in the transverse displacement from disease, the functions of the heart are not manifestly injured by the change of position.”†

In the case just detailed, if a suspicion had been entertained as to the cause of thoracic tympany, the crucial test of causing the patient to swallow some liquid, whilst the ear or stethoscope was applied to the left chest, might have been used, and the metallic tinkle of liquid entering the stomach within a few seconds after the visible movement of pharyngeal contraction, would have removed all doubt.

*Concentric displacement* of the heart consists in a movement of the organ *towards* the side whence the displacing force operates. Exception may be taken to this definition on the ground that the displacing force, atmospheric pressure, acts from without, and from all points of the circumference of the chest. This objection would be strictly valid. Nevertheless, the above admission having been expressly made, the impugned definition, which is found convenient, may be retained without prejudice to the interests of truth.

Concentric displacement is usually effected, not horizontally, but diagonally upwards and to one side. This is, no doubt, due to the fact that adhesion of the contracted lung to the cone of the pleura, has, in the majority of such cases, taken place pre-

\* *The Diseases of the Heart and Aorta*, 1854, p. 456.

† *Opus citat.*, p. 458.

viously to its recession. The causes of concentric displacement may be classified under two heads, namely,

1. Gradual decrease in the volume of the lung from intrinsic or substantive disease of the organ.
2. Compression of the lung by liquid or air in the pleura, and subsequent obliteration of its vesicular structure by simple condensation, or by constriction of false membrane; with removal of the compressing medium.

The causes which belong to the former category are these:

1. Tubercular atrophy of the lung, with or without cicatrization.
2. Cirrhosis of the lung.
3. Chronic gangrene of the lung.

Tubercular atrophy of the lung, as a cause of concentric displacement of the heart, has been well illustrated by Dr. Stokes, who explains the absence of cardiac displacement in ordinary phthisis by the usual presence of the disease in both lungs, and by the inflation of cavities in its advanced stages. The cases in which it is most likely to be presented are those familiar to every physician much conversant with thoracic disease, in which a slow process of wasting of the lung substance, accompanied with softening of small and distinct masses of tubercle, and formation of minute disseminated cavities, occurs. In such cases there is extreme flattening and retrocession of the anterior wall of the chest on one side, whilst the physical signs are those of small cavities dispersed throughout the lung.

Dr. Stokes dwells upon the fact that in many such cases cicatrization of the cavities takes place, and, in either view, and perhaps likewise in some measure from the comparative immunity of one lung, the patients not unfrequently survive many years, in a state of broken health, which ordinarily ends in death within as many months.

Many cases of this kind have come under my notice, and in most of them I have observed a proclivity to copious hæmoptysis. The patients sometimes cease to cough and actually recover flesh, especially on removal to the country; but the physical signs remain unaltered.

The direction of displacement from the cause last mentioned

is diagonally upwards, where, as usually happens, adhesion of the apex of the lung to the cone of the pleura has previously taken place. Whether displacement of the heart be due to traction of the retreating lung, as is generally held to be the case, or to atmospheric pressure, as I believe it is, the diagonal movement would be equally explicable, because in either case the receding chest-wall and the adjacent viscera would follow the retracting lung.

Cirrhosis of the lung, or dilatation of the bronchial tubes with condensation and atrophy of the pulmonary substance, and recently named "fibroid phthisis," by Dr. Andrew Clark, is the most frequent cause of concentric displacement of the heart. It operates in the same manner as chronic tubercular atrophy; but with this difference, that displacement is usually effected in the horizontal line, and because, as I believe, antecedent pleuritic adhesion to the apex of the pleura is not common.\* The diagnosis between the two last-mentioned causes will be found in many cases difficult, and must rest mainly upon the evidence of actual excavation upon the one hand, and upon the special symptoms and signs of cirrhosis on the other; amongst which I rank in the first place, a state of general and varicose engorgement of the superficial veins of the extremities, associated with great emaciation. Displacement of the heart, consecutive to the removal of air or liquid, may be best diagnosed by reference to the previous history, if such can be obtained from an accurate and reliable source. The displacement is always towards the sound side of the chest, in the first instance, and subsequently towards the affected side; whereas concentric displacement is primarily towards the affected side, and is permanent.

*Backward displacement* of the heart may take place as the result of tumors in the anterior mediastinum;† collections of air, pus, or blood in the same situation, or a posterior exostosis of

\* Dr. Hughes exhibited before the Pathological Society of Dublin, in the Session 1873-4, an example of cirrhosis of the left lung, with displacement of the heart to a point beneath the left clavicle. But in this case there were several vomices in the lung, which was adherent to the cone of the pleura.

† See Case 64 (A. Murray) "Cancer in Anterior Mediastinum."



the sternum. Normally, the point of apex-pulsation varies slightly with the position of the body. Thus, in the supine posture the apex recedes somewhat, and its impulse is less distinctly felt; in the prone position, on the contrary, it is brought by the weight of the heart into persistent contact with the anterior wall of the thorax; and in the lateral positions it gravitates about half an inch to the corresponding side.

As to the cause of the stroke of the heart against the side, on which the phenomenon of impulse depends, the most plausible, because apparently the most scientific, theory, is that first propounded by Sir James Alderson, and now known as that of Gutbrod. This is the recoil theory, and is identical with that by which the recoil of firearms, on being discharged, is explained. In the latter case, the recoil is due to the sudden derangement of equality of pressure on the internal surface of the arm at the moment of its discharge, which results from the absence of an opposing wall at the point of exit. The consequence is, that whereas at all other points centrifugal pressure is neutralized by equal pressure upon opposite points, the pressure made upon the surface opposite to, and equal in extent with, the orifice of exit, is not so neutralized, and tends to propel the entire body in the direction in which it operates. Unfortunately, however, for the ingenious authors of this theory, the simplicity of which so strongly recommends it to our acceptance, the parallel cannot be sustained; the recoil of firearms is in fact, and perceptibly, posterior in time to the explosion, as it must be if dependent upon the agency above explained; whereas the impulse of the heart is strictly synchronous and coeval with the first sound. I do not wish to deny that on the principle stated, a recoil of the apex must and does take place; but it is in the direction of the orifice of the aorta, *i.e.*, downwards, *backwards*, and towards the left, and in time perceptibly later than the first sound; it is the force by which the ventricles are elongated after systole, and the apex lowered to the position which it occupied previously to that act. It is well exemplified in pericardial frottement at the apex, which is usually characterized by a double rub, the first element of which coincides with the impulse and the first sound; and the second with the descent

of the apex, and is an abrupt, vigorous, and manifestly a "back" rub.

Dr. Sibson maintains that, during the impulse, the apex and the anterior walls of the ventricles move forwards by a lever movement, the fulcrum being the base of the pericardium attached to the central tendon of the diaphragm, and kept tense by attachment above to the investments of the great vessels. He says, the apex moves forwards and to the right, with a revolving movement upwards; there is a movement forwards and outwards, "the ventricles over their whole surface make a direct thrust against any object upon which they impinge, and wherever, therefore, they come in contact with the walls of the chest." The impulse is, therefore, in his opinion due to the impact, not alone of the apex, but of a great portion of the anterior surface of the ventricles. The cause of impulse he considers to be quintuple, viz.:

1. Muscular rigidity.
2. Lever movement of ventricles.
3. Outward pressure of blood.
4. Movement of repulsion (Alderson, Gutbrod, and Skoda).
5. Lengthening downwards of blood-column in aorta and pulmonary artery.\*

Pettigrew has shown that the fibres of the ventricles commence and end at the ventricular orifices; they therefore form elongated loops, the extremities of which are fixed at the base, whilst the looped portion, forming the apex, is free. Now, the direction of the heart's axis in a state of quiescence being downwards, forwards, and to the left, at an angle of about  $15^{\circ}$ , and the anterior wall of the chest forming a nearly vertical plane in front of it, the contraction of a group of such fibres, interlaced at the apex, must, by raising the apex towards their fixed points at the base, bring it into forcible collision with the anterior thoracic wall, at a point somewhat above the level of their loops in a state of repose. Hence the impulse of the heart.

*Double impulse*, as already stated, is associated with left ventricular hypertrophy of the excentric form. It is due to strong diastolic reaction of the left ventricle at the moment of inci-

\* *Medical Anatomy*, last fasciculus, 1869.

pient diastole, and coincides, therefore, with the period of ventricular recoil, according to Gutbrod's theory.

The ventricle being hypertrophied, and its cavity dilated, it is readily conceivable that, as the volume of blood entering it is in proportion, its reaction or recoil would involve a second impulse. Two forces are, no doubt, in operation here: the reaction of discharge as already explained, and the action of influx from the auricle at the moment of cessation of ventricular systole. Quite irrespectively of the explanation just offered, in regard to which difference of opinion is admissible, the phenomenon of double impulse is pathognomonic of dilated hypertrophy of the left ventricle, as yet in a state of tissue-soundness.\*

*The first sound* of the heart coincides with the impulse and the first two-thirds of ventricular systole; it is the product of a two-fold cause; namely, the impulse of the ventricles, and the sudden tension of the auriculo-ventricular valves and chordæ tendineæ. Other causes of sound, at the moment of ventricular systole, have been insisted upon; for example, the mutual friction of the fibres of the ventricles, giving rise to the muscular *susurrus*; the rush of blood over the rough surface of the ventricles, and through the arterial orifices; and the sudden raising of the sigmoid valves.

The last-mentioned cause cannot, in any degree, contribute to the first sound, because the only sonoric element of valvular movement, namely, valve-tension, is not involved in it. The valves are raised by a pressure *ab intra*, but are in no degree subjected to tension; hence, they can contribute no factor of systolic sound, unless by obstruction, which implies disease.

The sound produced by the movement of blood over a rough surface is certainly not of the character of the first sound of the heart, which seems to be a compound of a "thud" and a

\* Dr. Stokes has called attention (*Diseases of the Heart and Aorta*, p. 328,) to an impulse of a peculiar character, which is occasionally met with in cases of extreme softening and attenuation of the left ventricle; the impulse is diffused, weak, and flapping, and suggests rather the passive shoc of an aneurism than the active impulse of the ventricles. This observation I have often verified; it is of great diagnostic value.

“click,” the former, as I believe, caused by the impulse of the ventricles, and the latter by the sudden tension of the auriculo-ventricular valves and chordæ tendineæ.

The theory of muscular susurrus, as applied to the heart in explanation of its first sound by several writers, and recently advocated with so much ability by Professor Haughton,\* implies asynchronism of contraction of the fibres of the ventricles, as in voluntary muscles; otherwise, manifestly, intrinsic friction could not occur in the ventricular walls. But there is no asynchronism; the fibres of the ventricles contract simultaneously, and hence they develop no sound of interfascicular friction. The theory of mutual collision of the blood-corpuscles scarcely deserves notice; a sound produced by such a cause would be of a blowing character, as is the basic murmur of anæmia; but in health the first sound of the heart partakes in no degree of this character.

The duplex constitution of the first sound, and the twofold cause to which it is due, as above stated, viz., the impulse of the ventricles and the tension of the auriculo-ventricular valves, are, to my mind, satisfactorily proven by its occasional reduplication, to which special reference will be made further on; suffice it here to say, that reduplication of the first sound is the result of its resolution into the above-mentioned two elements.

Dogiel and Ludwig conclude from some experiments performed by them, that the first sound is due to the contraction of the muscular fibres of the ventricles exclusively, and that valve-tension has no share in its production. Dogs were poisoned with curara, the heart was exposed, and artificial respiration was maintained. The venæ cavæ, pulmonary artery, pulmonary veins, and aorta, were successively ligatured at their junction with the heart. The heart was then removed from the body of the animal, and placed in a glass vessel filled with defibrinated blood. By means of a peculiar arrangement, a stethoscope was made use of for the purpose of auscultating the heart within the vessel. As long as the heart pulsated, a first sound, but faint, was heard. They declare that previous to

\* *Outlines of a New Theory of Muscular Action*, 1863.



the removal of the heart from the body a similar result was obtained.\*

In reference to the foregoing, Guttman announces that he has repeated the experiments of Dogiel and Ludwig, with, to his mind, a contrary result. He declares that in the bloodless heart the systolic sound has a character quite different from that produced in the heart when the blood is flowing through it, and causing forcible closure and tension of the valves; but even in the bloodless heart, he maintains that valve-tension is not entirely excluded, inasmuch as when it contracts, the papillary muscles continue to contract and tighten the valves.†

There is much force in the foregoing remarks of Guttman, but in other respects the experiments detailed are singularly inconclusive. Deligation of the vessels connected with the heart, even if this were done in the order of the circulation, could not have been accomplished within the period of a single cycle of cardiac movement, so as effectually and completely to exclude blood from the heart; and if this were not effected, valve-pressure and tension became inevitable, so long as the contractions of the heart continued. Hence, the valvular element of sound cannot be regarded as excluded in these experiments.

*Reduplication of the first sound of the heart* may be exhibited in four different connections:

1st and oftenest, it is associated with simple functional derangement of the heart, as evinced by nervous palpitation and fluttering at the precordium (*vide* "Neurosis," Mr. Ralehan); variation in the rate of the pulse without irregularity; and flushings. There is likewise usually anæmia in some degree, and, in young females, leucorrhœa and cervical venous hum.

2nd. Attenuation and weakness of the ventricles, as exhibited in middle age by those of a nervous and fretful temperament, and evinced by feeble cardiac action with or without irregularity, and sharp clear sounds.

\* *Ludwig's Arbeiten*, 1868. Abstract by Hermann in *Centralblatt*, No. 31, 1868; and *Dublin Quarterly Journal of Medicine*, November, 1869.

† *Virchow's Arch.*, xlv., p. 223; also *Journal of Anat. and Physiology*, No. V. (November, 1869), p. 168.

3rd. Weak and softened heart in process of tissue-degeneration, with dilated and atheromatous arteries.

4th. Ventricular hypertrophy of the *simple* form.

The attempt to explain the phenomenon of reduplication of the first sound, in any of these conditions, would involve speculation in the region of the unknown. In the first three there is, presumably, derangement either of synchroniety in the influx of nerve-force, or of muscular irritability in the walls of the heart. It is conceivable that, owing to one or both of these causes, the contraction of the papillary muscles, and, therefore, the tension of the tendinous chords and the valves, may be perceptibly later in occurrence than that of the ventricular walls. Such derangement would not, of necessity, involve retroversion of the valves and reflux of blood, because, by the pressure of the mass of blood, the valves are brought into mutual apposition at the first moment of ventricular contraction, whereas, valve-tension and sound are the joint product of parietal and papillary contraction. In the former of these periods the active pressure upon the valves is comparatively insignificant, and the mere extension of the papillary muscles and tendinous chords suffices to prevent their retroversion; but, during the latter period, corresponding to the acme of ventricular systole, the active contraction of the papillary muscles is requisite to obviate that result. Of its occurrence in simple hypertrophy, an explanation will be given further on.

Reduplication of the first sound I therefore regard as equivalent to the resolution of it into its two constituent elements, the ventricular impulse, and the click of valvular tension; accordingly, the former element of the double sound should be of a dull and muffled character, and the latter sharp and clear. Such is actually the case, according to my experience, without an exception.

*Suppression* of the first sound may occur in either of the following conditions, viz:

1. Adipose softening of the heart.
2. Typhous softening of the ventricles.

Loss of the first sound, in the former of these conditions, is the result in greatest part of the enfeeblement of the ventricles by

structural deterioration, by which both the impulse of the ventricles and the tension of the valves are impaired; it is, however, in some degree likewise due to imperfect conduction of sound through the softened walls of the ventricles. It will be hereafter shown that suppression of impulse and of first sound, in association with a certain group of symptoms having reference chiefly to the encephalon and the pulse, warrants the diagnosis of fatty heart.

Louis has called attention to suppression of the first sound of the heart in enteric fever, as indicative of softening of the ventricles.\* Dr. Stokes subsequently pointed out this sign as peculiar to typhus fever from the sixth to the fourteenth day; and in typhus, and when not previously exhibited, as pathognomonic of ventricular softening. Most frequently the left ventricle only will be found in this condition; occasionally both ventricles are engaged, but the left in a greater degree than the right; and still more rarely the entire organ is similarly affected. The condition adverted to is not the result of putrefactive change; it is strictly pathological, non-inflammatory, and specific in character. Although indicative of so grave a structural lesion of a vital organ, this sign is not usually associated with a fatal issue of the disease, and constitutes an indication for the free administration of wine.†

*Association of phenomena with the systole of the ventricles.* Concurrently with the commencement of ventricular systole, as already stated, the impulse and the first sound are delivered, which imply the impact of the apex against the chest wall, and the closure and tension of the auriculo-ventricular valves.

Preparatory to the impulse, as pointed out especially by Professor Halford,‡ the base of the ventricles descends, whilst the apex ascends, but to a less extent, and the central portion expands or attains increased bulk. By the descent of the ventricles space is afforded for the diastole of the auricles, and a point of resistance is given to the aorta and pulmonary artery, which are about to be put upon the stretch by the vigorous

\* *Researches on Gastro-Enteritis*, Bowditch's translation, vol. i.

† *Dublin Journal of Medical Science, and Diseases of the Heart and Aorta*, p. 370.

‡ *The Action and Sounds of the Heart*, 1860.

injection of blood from the ventricles. The blood is driven with great force into the aorta and pulmonary artery, which are distended to their uttermost capacity, and the branches arising from the former of these vessels are filled consecutively in the order of their origin from the main trunk. Thus, the carotid and subclavian arteries are supplied and pulsate almost simultaneously with the impulse of the heart, but the radials perceptibly later than the impulse.

Dr. Burdon Sanderson says the radial pulse is one-sixth of a second later than that of the carotids, owing to the elasticity of the large arteries; where these are unyielding there is no postponement of the peripheral expansion. He says the heart exerts its greatest vigour at the commencement of ventricular systole, in forcing open the sigmoid valves. The rate of transmission of the percussion wave in the arteries he calculates at about 90 feet per second. The bursting open of the aortic valves causes a sensible vibration, which may be mistaken for ordinary arterial expansion (?), but it reaches the peripheral arteries in about 1-50th of a second.\*

During the contraction of the left ventricle, according to Sibson, "the cavity changes its place, being more to the right, and with its axis, which was formerly in the direction of the auricle, now pointing to the aorta. The contraction proceeds in a twisting manner; the blood is, as it were, wrung out of the cavity, and with a current that takes naturally the twisted direction of the spring and arch of the aorta.†

"The cavity of the ventricle, which at the end of the diastole is somewhat egg-shaped, is totally changed in form at the end of the systole. It is then narrow and triangular, except towards the apex, where it is obliterated; and it presents a spiral twist, in a direction from the aortic orifice to the apex. This spiral course commences at the aortic valves, where it bears backwards and from right to left, is just appreciable in the body of the cavity, and becomes again marked towards the apex, where it bears forwards and from left to right."‡

\* *Lectures delivered at University College, Lecture vi. ; Medical Times and Gazette, April 29, 1871.*

† *Prov. Med. Transact., 1844, p. 518.*

‡ *Medical Anatomy, last fasciculus, "Movements and Sounds of the Heart."*



The blood thus escapes from the ventricle in a succession of spiral curves, by which, as in the rifle, precision of movement is insured, and the effects of friction are, to a certain extent, obviated. The spiral fibres of the walls grasp the papillary muscles and fleshy columns "like the coils of a serpent," forcing them together and expelling the blood. The mitral portion of the ventricle is entirely obliterated by the packing of the papillary muscles and fleshy columns, leaving for the passage of the blood only the small spiral channel already described, as leading in the direction of the aorta.

In relation to the closure of the mitral valve in systole, a space of much interest has been described by Sibson, under the name of "intervalvular space," into which "the aortic valves, during diastole, play directly;" and owing to the existence of which "the mitral valve is closed up to the end of systole, by the pressure of the blood on its anterior flap." "Its immediate walls are everywhere rigid and aponeurotic, and it cannot, therefore, be compressed by the contraction of the muscular walls during the systole." This space is described as being bounded in front by the aortic valves, behind by the anterior segment of the mitral valve, to the right by the central fibro-cartilage or tendinous septum, and externally or to the left by the muscular wall of the ventricle, "which is here lined by a rigid aponeurosis."\*

He says that, in contraction, the left ventricle forms a shoulder round the mitral valve, which, however, it never touches. The direction and range of movement of the walls of the several chambers of the heart, and the relative direction and course of the several currents of entrance and exit, are admirably represented in the annexed engravings (Figs. XVII and XVIII), borrowed from Dr. Sibson.

Dr. Sibson refers to these engravings in the following terms: "In order that the successive movements of the heart might be observed with accuracy, a millimetre measure was stretched across the organ, as in Fig. I [XVII], and its precise position in relation to the measure was noticed, first at the end of the diastole, and then of the systole." "In those figures [XVII and XVIII] the

\* *Medical Anatomy.*

FIG. XVII.

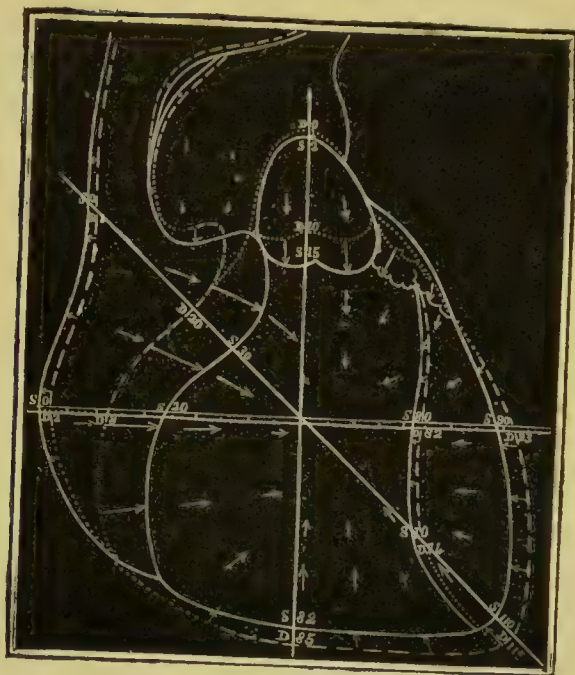


FIG. XVIII.

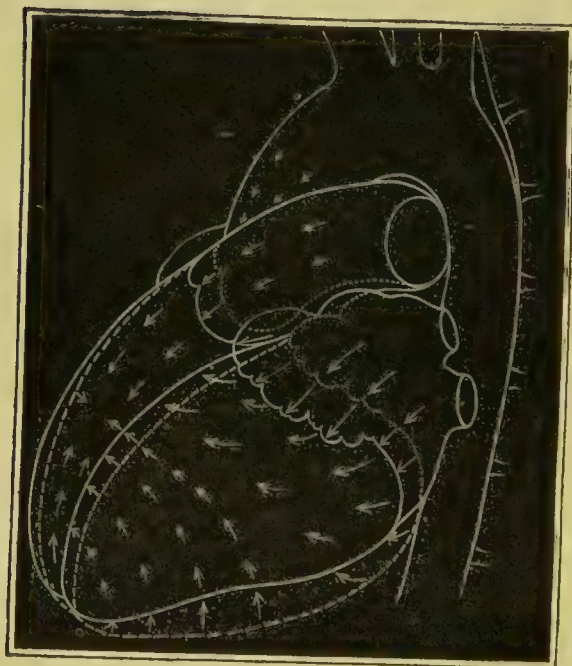
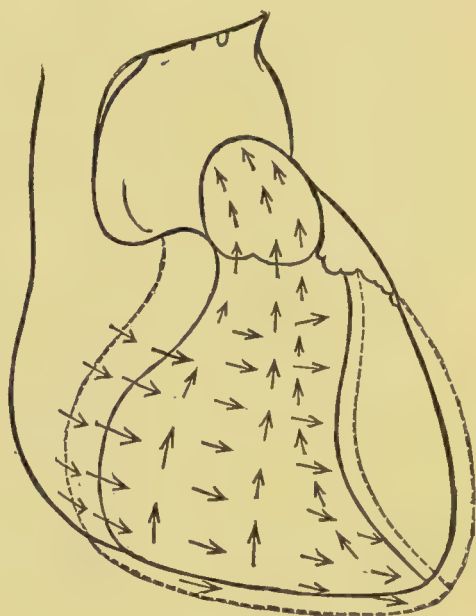


FIG. XIX.



FIG. XX.



outlines of the cavities of the heart and the great vessels are indicated, at the end of the systole of the heart by continuous lines; at the end of the diastole by interrupted or dotted lines. The dotted are less accurate than the interrupted lines. The systolic movements are everywhere shown by arrows, the extent of the movements being marked by the length of the arrows. It is more difficult to watch and measure the movements of the heart at the side than at the front. The side view, Fig. II [XVIII], is, therefore, not so accurate as the front view, Fig. I [XVII]. I believe, however, that both figures very fairly represent the movements of the heart.

“In the left ventricle, the aperture of entrance through the mitral orifice is contiguous to the aperture of exit at the aorta, the two orifices being separated by a membranous septum. In the right ventricle the aperture of entrance through the tricuspid orifice is at a distance from the aperture of exit at the pulmonary artery, the two orifices being separated by the muscular channel of the *conus arteriosus*. In the left ventricle the current of blood inwards, which descends during diastole behind the anterior segment of the mitral valve, is parallel in direction to the

current of blood outwards, which ascends during the systole in front of that segment [Fig. XIX annexed]. In the right ventricle the current of blood inwards is at right angles to the current of blood outwards, since the blood enters the cavity from right to left, and leaves it from below upwards [Fig. XX annexed]. During the systole the stream of blood in the left ventricle takes a spiral direction towards the aortic orifice, in accordance with the spiral direction of the aorta itself [Fig. XIX]. The stream of blood in the right ventricle, as it ascends, mounts over the bulging septum, being restrained by the concave free walls. This upward stream, which narrows as it proceeds, thus takes the curved direction upwards, backwards, and inwards, of the *conus arteriosus* and the pulmonary artery. In the left ventricle the anterior segment of the mitral valve, and the right and left papillary muscles, form a hollow channel for the stream of blood, which, as it ascends to the aorta, presses upon the under surface of the valve. In the right ventricle the stream of blood, as it ascends, sweeps onwards at right angles to the under surface of the tricuspid valve, and rushes between and across the papillary muscles, and through the tendinous cordage that connects those muscles to the valve.”\*

I am convinced that the relative position of the papillary muscles and fleshy columns on the inner surface of the ventricle is such that, at the acme of systole, the former pass mutually to opposite sides of the chamber, and so by crossing lines of tension stretch the valves to the uttermost, and thus oppose to the escaping blood at the acme of systole, the maximum of resistance.† Sibson says the tip of the anterior right papillary muscle contracts towards the septum, and those of the left approach each other, and both extremities are approximated to the “zone of rest, or stable equilibrium.”

*The short, or systolic pause*, equal in duration to one-third of the systole, immediately succeeds the impulse and the first sound. During this brief period of silence the ventricles are still in contraction, and blood is in forced and rapid efflux through the aorta and pulmonary artery, the auriculo-ventri-

\* *Opus citat.*, p. 13.

† See a paper by the author in the *British Medical Journal*, December, 1867.



cular valves being closed, and the sigmoid valves raised. If the ventricles were not still in contraction, the second sound, being determined by arterial reaction, would immediately succeed the first. On the other hand, it is no less clear that if ventricular systole persist through the short pause, the first sound, if in any measure due to the mutual friction of the fibres of the ventricles in contraction, or to the attrition of the escaping blood against the walls of the ventricles and arteries, should be likewise, in some degree, continued through it. That no sound is audible, however, through one-third of the period of ventricular systole, is to my mind proof that neither of these alleged causes of the first sound contributes in any measure to its production.

The force actually exercised by the heart in contraction is a problem of great interest, but the solution of it is attended with much difficulty, partly from the abstruse nature of the inquiry itself, but in a great degree also from the number of minor problems involved in it.

Hales calculated from his experiments that the hydrostatical pressure within the hearts of oxen and horses amounts to nine feet perpendicular of liquid blood.

Dr. Haughton estimates that in the human heart at 9·923 feet; and calculates that each ounce of human heart-muscle is capable of lifting a weight of 20lbs. through the height of one foot every minute; and finally, that in health, a propelling force equivalent to this is actually in operation throughout our lives.\*

The *second sound* terminates the short or systolic pause; it is sharp, clear, and abrupt; loudest at mid-sternum, and at the sternal end of the left second intercostal space. Around each of these centres it is diffused through a radius of about two inches in the adult male, but with greater distinctness in three lines of direction than in others, viz., downwards and to the left towards the apex; vertically downwards to the xiphoid cartilage; and upwards to the right second costo-sternal junction.

The second sound is much more extensively diffused in females and in children, because in childhood and in the female sex it is of a sharper and clearer quality, and the thoracic frame-

\* *Lectures on The Principle of Least Action in Nature*, Lecture III.; *British Medical Journal*, June 17, 1871.

work in these subjects is a better conductor of sound. I have repeatedly heard the second sound, and the first still more distinctly, under both clavicles, and extensively on either side of the back, in nervous females. In children this is the rule, and absence of the cardiac sounds at any point of the chest, the rare exception.

As to the *cause* of the second sound, there is now no second opinion amongst writers of authority. It is the product of the sudden closure and tension of the sigmoid valves in the aorta and pulmonary artery, under the elastic reaction of the walls of these vessels, consecutive to ventricular systole. The contractile force of the ventricles is in great part stored up or rendered latent in the walls of these vessels, to be restored as reacting force, by which the vessels resume their normal diameter at the end of ventricular systole, propelling the contained blood onwards, and so completing and sustaining the wave of pulsation; but likewise repelling it in the direction of the ventricles, and thus closing the sigmoid valves with an audible click, constituting the second sound. The two sounds of the heart may be therefore contrasted in regard to quality, duration, constitution, cause, and mode of origin as follows, viz. :—

		<i>First sound.</i>	<i>Second sound.</i>
Quality	...	Dull.	Clear.
Duration	...	Prolonged.	Abrupt.
Constitution	...	Compound.	Simple.
Cause	...	Impulse, valve-tension (in part active).	Valve-tension (entirely passive).
Mode of Origin	...	Direct and immediate result of ventricular contraction.	Indirect and remote result of ventricular contraction.

*Reduplication of the second sound* is of much more frequent occurrence than reduplication of the first. It is most frequently met with in association with presystolic murmur, and may be therefore regarded as a *presumptive* sign of mitral obstruction.

Next in frequency of occurrence is the combination of double second sound with valvular inadequacy at the aortic orifice. In all such cases there is, of course, a murmur in association with one of the two elements of the double second sound, and this element is invariably the last. (Cases of Hutchinson, and Mrs.

Ewell.)\* The existence of a murmur of aortic reflux in union with the second element, renders it easy to identify this latter as aortic in origin. In all such cases the left ventricle will be found dilated ; hence the evacuation of that chamber occupies a longer period, and the reaction of the aorta is postponed in an equal ratio, and rendered posterior in time to the corresponding phenomenon on the right side of the heart. Conversely, when reduplicated second sound exists in connection with disease at either arterial orifice, it may be regarded as evidence of dilatation of the corresponding ventricle. The cause of reduplication of the second sound is not, as has been alleged, derangement of synchronism in the closure of the segments of either sigmoid valve. If such were the cause there should be a murmur of reflux always associated with the reduplicated sound, but this is notoriously not the case in the majority of examples of it. Again, I have repeatedly made the following observation, which seems conclusive as against the alleged univalvular origin of double second sound. Whilst a double second sound was equally audible at the orifice of the aorta and that of the pulmonary artery, but most distinctly so at a point intermediate to and equidistant from both, a single second sound only existed at the distance of an inch or so external to either orifice in the horizontal line. Manifestly, if derangement of synchronism in the closure of the segments of either set of valves had been the cause of reduplication, the latter would have been audible to an equal distance from the orifice in the directions inwards and outwards, and it would have been most distinct at the orifice itself, and not midway between the two orifices. The crucial test, however, is supplied where diastolic murmur exists at the aortic orifice ; in that case a second sound *and* a murmur are audible in the situation of both orifices, and at all points intermediate to them ; but over that of the aorta the murmur preponderates, and is posterior in time, whilst the normal sound is in excess over the valves of the pulmonary artery.

Where the second sound is reduplicated in connection with mitral stenosis, the element deranged is most likely that produced in the pulmonary artery, the entire pulmonary system,

\* *Vide* "Aortic Regurgitation" and "Mitral Obstruction."

and the right chambers being engorged by obstruction at the mitral orifice. In the effort to overcome this obstruction, the systole of the right ventricle is protracted, and the reaction of the pulmonary artery proportionately postponed. The reaction of the aorta is, on the other hand, in mitral stenosis most probably anticipated, where the left ventricle is reduced in capacity, as always is the case where mitral or aortic reflux does not co-exist. Hence, it is likely that in simple mitral stenosis two causes of doubling of the second sound are in operation; namely, diminished capacity of the left, and dilatation of the right ventricle.

I would, therefore, contrast the two conditions under which re-duplication of the second sound may be witnessed, as follows:

*In Aorta.*

1. Second sound, accompanied or replaced by a murmur, and postponed.
2. Second sound, normal as to quality, but premature in occurrence.

*In Pulmonary Artery.*

1. Second sound, normal as to time and quality.
2. Second sound postponed, but normal as to quality.

If the preceding observations be well founded, and I believe they are, it follows:

1. That dilatation of *either* ventricle must be accompanied by doubling of the second sound.
2. That the side of the heart at which the *postponed* element of the double second sound takes origin, is that of the dilated ventricle.
3. That equal dilatation of *both* ventricles will be unattended with doubling of the second sound.
4. That the arrest of reduplication of the second sound may be due either to improvement in the condition of a ventricle previously dilated, or to dilatation of a ventricle previously normal in capacity.
5. That, generally, the causes of reduplication of the second sound are those which give rise to difference in the capacity of the two ventricles.

I cannot agree with Dr. Richardson in the opinion that reduplication of the sounds of the heart is rare.\* I have met with and noted it in seventeen instances: viz., in ten there was

\* *Clinical Essays*, 1862.



reduplication of the first sound ; in six, of the second sound ; and in one, of both sounds.\* Nor can I admit his assumption, to the effect, that a third short sound, taking the place of the pause, is frequently mistaken for a reduplicated sound. A short sound of this character, corresponding to what I shall have to describe as an "aborted" sound, is characteristic of failing heart, and *always* irregular in its recurrence ; whereas reduplication is singularly regular. He has not met with an example of doubling of the first sound, of which the majority of my cases are examples ; and has met with only three cases of doubling of the second sound. His explanation of the phenomenon is, that it arises from asynchronism in the closure of the semilunar valves, consequent, as he believes, upon pulmonary obstruction, prolongation of the first sound, and retardation of the second sound on the *right side*.

Giegel and Guttemann coincide in the opinion just stated as to the cause of reduplication of the second sound. They have met with it pretty frequently in mitral narrowing, with or without regurgitation. Most of the patients were thin and anæmic, and had been long the subjects of heart-disease. The double sound was audible over the tricuspid, aortic, and pulmonary orifices. These writers regard doubling of the second sound, when constant, as diagnostic of stenosis or inadequacy of the mitral orifice.† It can, however, equally exist in all cases of stasis or engorgement of the right side of the heart, or of dilatation of the pulmonary artery ; but in these latter cases the doubling would not be so constant as where valve disease exists.

It may be also observed in young tuberculous subjects ; in those suffering from pulmonary emphysema ; in pleurisy ; and in fatty degeneration of the heart.

Guttemann, however, does not agree with Geigel in the opinion, that doubling of the second sound is a *constant* sign of mitral constriction ; and in his dissent I fully concur. He regards the connection as rare, and present only when the patient

\* In several instances, not included in this summary, I have observed doubling of the second sound in connection with the dilated right ventricle of pulmonary emphysema, with intercurrent or chronic bronchitis.

† *Archives Générales de Médecine*, June, 1869.

is in a state of complete repose. This, however, is by much too exclusive.

*Suppression of the second sound*, I have witnessed only in the stage of collapse of Asiatic cholera. I believe I was the first to call attention to this negative phenomenon in cholera, in the report of the cholera epidemic of 1866, as witnessed in the Mater Misericordiæ Hospital, and published conjointly by myself and Dr. Cruise. Dr. Henry Kennedy of Dublin would seem to have observed it also in the same epidemic, as he announced his notice of it, and quite irrespectively of mine, in the discussion which followed the reading of the several cholera reports, at the College of Physicians in June, 1867. In the collapse of cholera the circulation is arrested primarily in the branches of the pulmonary artery, and retrogressively in the trunk of that vessel. Hence the twofold effect of *depletion* of the left ventricle and aorta, and *repletion* of the pulmonary artery and right ventricle. In the extreme of this condition it is conceivable how, from the opposite causes above mentioned, failure of re-action in the aorta and pulmonary artery, and therefore of the second sound, might ensue. Irrespective of the value of the foregoing hypothesis, and whatever the true explanation may be, the fact is unquestionable, that in the last stage of cholera, and, as far as I am aware, in that condition exclusively, complete suppression of the second sound may be observed, as of almost constant occurrence.

The observations of Dr. Stokes on the modification of the impulse and the sounds of the heart in typhus fever, are of the utmost value and importance, and will find a suitable place here.

A jerking impulse, with clear sounds, in the advanced stage of typhus, should be regarded with much apprehension of a fatal issue.

Partial or total loss of impulse, in connection with suppression of the first sound on the left side exclusively, or on both sides of the heart, has been repeatedly observed from the eleventh to the fourteenth day of typhus, but it has been met with as early as the seventh day. In these cases one or both sides of the heart, according to the extent of suppression, has been found

soft, friable, and livid, with a viscid exudation between the fibres. In one remarkable example (Case 48) not only the impulse, but both sounds also, were lost on the twelfth day of fever; the heart was found to be soft and flabby, spreading out upon the table by its own weight, and when held up by the great vessels, resting on the closed hand, it fell over the latter like an inverted cup. The order of modification of the sounds, he has observed to be as follows:

1. Diminution of first sound over left ventricle.
2. Diminution of first sound over right ventricle.
3. Cessation of first sound over left ventricle.
4. Cessation of first sound over right ventricle.
5. Return of first sound over right ventricle.
6. Return of first sound over left ventricle.

He has observed, likewise, a foetal character of both sounds, which, nevertheless, were distinct, the pulse being over 130; and he has remarked that in some cases of typhus where the first sound was lost, the radial pulse continued, and even exhibited average strength and volume. The radial pulse has continued thirty-six to forty-eight hours after all trace of impulse and sound had disappeared. From the seventh to the fourteenth day of a case of typhus the first sound and impulse were nearly lost, whilst the second sound was very loud, and a second and strong impulse existed.\*

*The diastole of the ventricles* commences with the second sound, and terminates only with the next succeeding impulse and first sound. The cause of the initiation of diastole has been by some writers considered to be the impetus of the blood propelled into the ventricle by the auricle during the contraction of the latter cavity. In refutation of this doctrine it is sufficient to observe, that active contraction of the auricles takes place only at the *end* of ventricular diastole, and immediately previous to ventricular systole. Longet maintains that the movement of ventricular diastole is due to the two-fold cause of the elasticity of the walls of the ventricle, by which, after contraction, they tend to separate, and thus attain a state of equilibrium, as does a caoutchouc bottle; and the influence of a virtual void in the

\* *Diseases of the Heart and Aorta*, p. 328, *et sequent.*

chest, created by the retraction of the lungs in expiration.\* I believe the ventricles, especially the left, possess normally an intrinsic power of expansion in virtue of their elasticity, but I also hold that this intrinsic power is materially supplemented by the extrinsic force of auricular reaction and contraction, operating through the medium of a large mass of blood, the mere weight of which contributes in some degree to the expansion of the ventricles. It should be remembered that ventricular diastole *cannot* in health coincide with expiration oftener than once in every four pulsations of the heart, and it *may* not even so often. Dr. Garrod maintains the existence of an active diastole of the ventricles, dependent upon the active turgescence of the walls of the heart, "consequent on the flow of blood into the coronary arteries immediately after the systole," "the auricles, from their thinness, not being similarly affected." He endeavoured to prove this by injecting water backwards into the aorta of a sheep's heart. He further concludes that, during ventricular diastole, there is in operation an absorptive force or attraction towards the ventricles.† Independently of the objection that, under the influence of the same agency, the auricles are supposed to contract and the ventricles to dilate, it must be remembered that in Brackyn's experiments the alternate action of the auricles and the ventricles was maintained without a coronary circulation of any kind.

The duration of diastole is equal to two-thirds of the entire cycle, and it includes three important events; namely, the second sound previously discussed; the long pause; and the acme or momentum of auricular systole. (See Fig. XVI.)

*The long or diastolic pause* is equal in length to the diastole, *minus* the second sound. It is a pause only in the sense that no movement of the heart, audible or palpable through the walls of the chest, takes place during its continuance; but in no other.

At the commencement of diastole, and coincidently with the second sound, the auriculo-ventricular valves are thrown open, partly in consequence of the removal of pressure from their

\* *Traité de Physiologie*, 3ième edition, 1869, tome ii. p. 132.

† *Journal of Anatomy and Physiology*, No. iv., May, 1869.



ventricular aspect, which occurs at the cessation of ventricular systole, but mainly owing to the pressure on their auricular surface, of the mass of blood which had been accumulating in the auricles during the systole of the ventricles, and which now, under the reaction of the auricular walls, forces open the valves and enters the ventricles at a gush.

An extreme degree of mitral narrowing, or other form of aggravated obstruction at the mitral orifice, may give rise to a *quasi* mitral diastolic murmur, but such murmur is really post-diastolic in time.\*

A true apex diastolic murmur may arise from aneurism of either ventricle of the heart, *and from this only*. Both the cause and the phenomenon are of extreme rarity. I cannot recall more than one example of the latter. (Case 68, James Toole.)† Dr. Sibson is of opinion that the auriculo-ventricular valves are raised in ventricular diastole by the papillary muscles, and "when diastole is complete the anterior flap (of the mitral valve) is held tight between the two papillary muscles."‡ Notwithstanding the high authority of Dr. Sibson I doubt the correctness of this view. I incline to think that if such were the agency by which the auriculo-ventricular valves were raised in diastole, and if, at its acme, they were thus held open and fixed by unyielding tendons and extended muscles, at the first moment of ventricular systole reflux into the auricle and systolic murmur would inevitably occur. On the contrary, I believe that the papillary muscles and attached tendinous chords, in a state of complete relaxation, and not hardened by immersion in spirit, are of sufficient length to admit of the valves closing over the orifice, even in the state of complete diastole of the ventricle. Blood entering the ventricle from the auricle first displaces the valve, pressing its segments against the walls of the ventricle; in proportion as the ventricle becomes filled, blood accumulates between its walls and the ventricular surface of the valves, and the latter are by this pressure displaced, and *floated* towards the auriculo-ventricular orifice. During this entire period, commencing with the second sound, and extending through the greater portion of the long pause, the auricles are in a state of undu-

\* *Vide* "Mitral Obstruction." Cases of Anne Coates and Mary Brennan.

† *Vide* "Aneurism of Heart."

‡ *Opus citat.*, p. 17.

latory or vermicular contraction, setting in from the appendix in the direction of the ventricle. By this movement of the auricles, the blood which enters them from the great veins is passed as quickly into the ventricles, but without the pressure of active muscular contraction. At the close of diastole, however, and therefore at the end of the long pause, the auricles having attained, in the language of Cruveilhier, a *momentum*, or condition of nerve-force most favourable to contraction, compress their contents with vigour, and propel the blood into the ventricles. These chambers, being thereby brought into a state of maximum distension, and receiving the stimulus to active contraction which that state supplies, contract in their turn, and so complete the cycle by initiating another act of ventricular systole. The long pause therefore commences after the second sound, and terminates at the first sound; and the movement of active auricular contraction which I have designated the *momentum of auricular systole*, concludes the long pause, and completes the diastole and distension of the ventricles. (See Fig. XVI.) It is readily conceivable that obstruction to the entrance of blood from the auricle would, at this period, when the other condition necessary for the production of bruit is supplied, namely, maximum pressure from behind, be most likely to give rise to murmur; and such is actually the case, as witnessed in the production of the pathognomonic sign of presystolic bruit.

Longet admits the two pauses, and describes the first "as a slight interval between the contraction of the auricles and that of the ventricles;" and in reference to the second he says, "after contraction of the ventricles the entire heart is relaxed or in a state of repose, during which it fills."

In regard to the relative effect on the two pauses, of variation in the rate of pulsation, Dr. Burdon Sanderson remarks:\* "The closure of the aortic valve was followed by a diastolic wave of great intensity, with reference to which it was interesting to notice that in this, as in all other instances in which I have had occasion to make the observation, the pyrexial acceleration of the pulse was not attended with any change in the duration of

\* "A Lecture on the Characters of the Arterial Pulse, &c.," part ii.; *British Medical Journal*, 20th July, 1867.

the systolic period, that is to say, the heart beat more frequently, not because its movements were more quickly performed, but it took a shorter time to repose between them."

In a paper read by me before the Medical Society of the College of Physicians of Ireland, in May, 1865, I stated as follows: "In regard to the effect of diminished rate of pulsation on the rhythm of the heart, it would appear that the diastolic pause is *relatively*, as well as absolutely prolonged thereby. I have had for some time past, and still have under observation in hospital, a case of permanently slow pulse; it is that of a man aged seventy-five, with œdema of the feet and other symptoms of weak heart. The pulse is perfectly regular, and in the recumbent posture only *thirty* in the minute. The heart's action is equally regular. The sounds are somewhat dull, but unattended with murmur. In this case, therefore, the heart pulsates once in every 2", of which  $1\frac{1}{2}"$  ( $1\frac{1}{3}"$ ) less the duration of the second sound is occupied by the diastolic pause, and the remaining  $\frac{1}{2}"$  ( $\frac{2}{3}"$ ) by the first sound and systolic pause." "As to the connexion between the radial pulse and the movements of the heart, it is commonly supposed that the radial pulse succeeds the first sound, and comes between that and the second sound. This, however, is true only when the pulse is at or near its normal rate. When the rate is under 126 the stroke of the radial artery corresponds to the systolic pause; but if the pulse amounts to 126 in the minute, it will be found that the beat of the radial artery is shifted back in the order of phenomena, and is now contemporaneous with the second sound. If the heart's action be still further accelerated, it will be found that the radial pulse is postponed in the same proportion, and now falls within the period of the diastolic pause." \*

I believe the opinion stated in the foregoing extract to be substantially correct; and with the qualifications as to time included within brackets, and without binding myself absolutely to the rate of pulsation given above, I still adhere to it in its entirety.

The pre-systolic contraction of the auricles is spoken of by Sibson as taking place "just before the ventricular systole,"†

\* "On the Rhythm and Sounds of the Heart," *Dublin Quarterly Journal of Medicine*, (Vol. xl., No. lxxx, New Series.)

† *Opus citat.*, p. 4.

and Marey, in Fig. X. of his great work\* represents the auricular systole by a rather abrupt ascent in the tracing, as preceding the systole of the ventricles by about one-tenth of a second. (See Fig. XXI.)

*Accentuation* of the second sound has been noticed in connection with three several conditions, viz. :

Disease at the mitral orifice,

Aneurism of the arch of the aorta,

Dilatation and atheroma of the arch of the aorta.

Skoda is of opinion that disease of the mitral valve, obstructive or regurgitative, is invariably accompanied by accentuation of the second sound in the pulmonary artery, and impairment or partial suppression of that sound in the aorta.† The explanation offered of this phenomenon is, that in such case the right ventricle, being of necessity hypertrophied, would, by its systole, dilate the pulmonary artery with increased force, and thereby induce a proportionate reaction in that vessel; whilst the volume of blood entering the aorta, being reduced, would dilate that vessel but imperfectly, and thereby induce feeble reaction and faint second sound in the aorta.

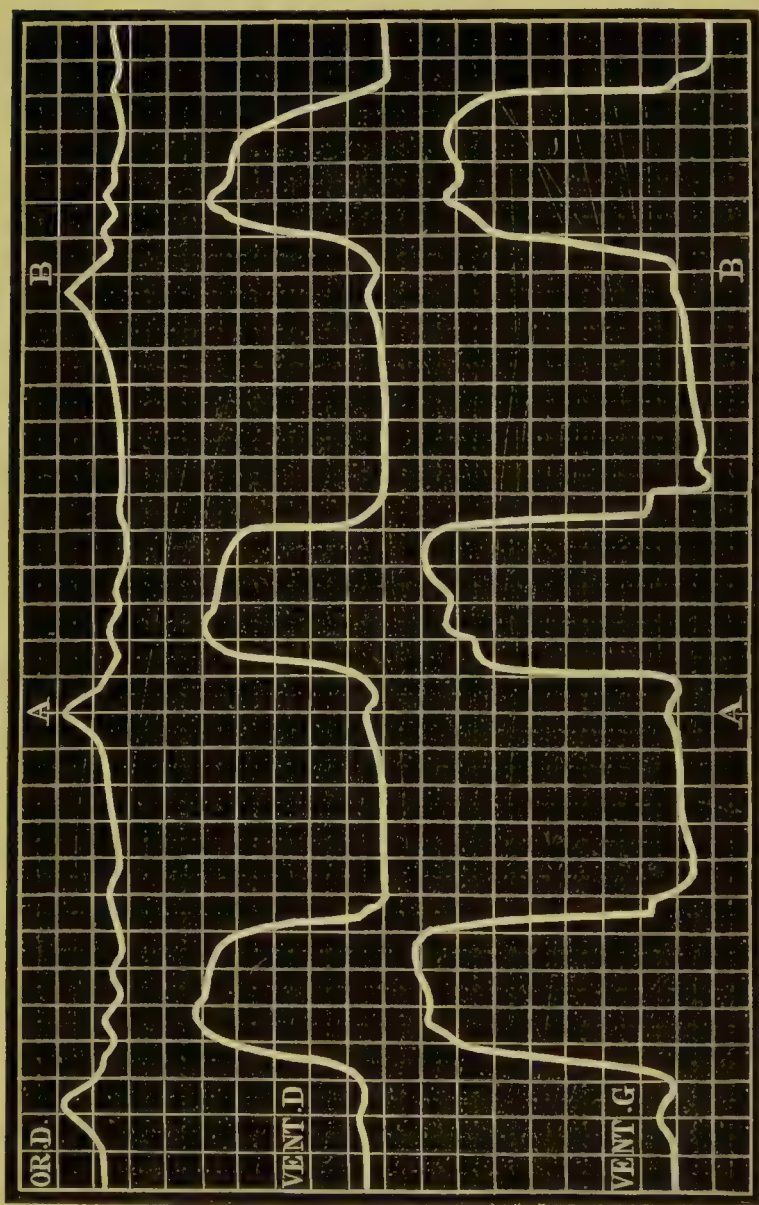
Manifestly the organic changes necessary for the production of these secondary phenomena are of late occurrence in mitral disease; nor is right ventricular hypertrophy always associated with it. In many cases the right ventricle is found to undergo simple dilation, without increased parietal development. These are cases in which tissue change of a degenerative character is in actual progress in the right ventricle, and the same cause which prevents hypertrophy, precludes likewise its consequence, accentuated second sound. Whatever the explanation may be, I am safe in asserting that not one-half the cases of indubitable disease of the mitral valve which have come under my notice, have presented the sign of accentuated second sound in the pulmonary artery. This sign is, however, manifested in very different numerical proportion in the two forms of mitral disease. It is much oftener associated with narrowing of the mitral orifice, than with inadequacy of its valves.

\* *Physiol. Médic. de la Circulation du Sang.*

† *On Auscultation*, by Markham, p. 334.



FIG. XXI.  
[From Marey, *Fig. 10.*]



Dr. J. Warburton Begbie\* calls attention to an "accentuated condition, or booming character" of the second sound, in aneurism, and in dilatation of the aorta; he has found that, "excluding the accentuated pulmonary second sound, and the intensified aortic second sound in some cases of hypertrophy and dilatation of the left ventricle, the accentuated second sound in the aorta is an indication of aortic aneurism, or of dilatation of the aorta associated with atheromatous degeneration." "The aneurism probably does not arise within the pericardium, and probably does not affect the ascending portion of the arch, but has most likely its seat in the transverse portion."

He explains this by reference to "increased recoil of blood on the valves, owing to diminution or destruction of the support given to the circulation by the artery."

Morbid condition of the valves, thickening and patchy hardness of them, may heighten or intensify the sound; and increased calibre of the vessel, may likewise, in Dr. Begbie's opinion, have the same effect.

Finally, he says that the sound is loudest over the aortic valves, both in aneurism and dilation of the aorta, but louder in the latter than in the former over the manubrium sterni.

Whilst fully admitting the deservedly high authority of Dr. Warburton Begbie on this and all other kindred subjects, I feel bound to declare that I cannot agree with him in the opinion, that accentuated aortic second sound is associated with aneurism of the arch, in sufficient numerical proportion to warrant its being regarded, even with the reservations mentioned, as a diagnostic sign of that disease. Its presence, in connection with thoracic aneurism, has certainly been, in my experience, the exception rather than the rule. The case is, however, different in regard to dilatation and atheroma of the aorta. In the absence of disorganization or inadequacy of the aortic valves, and associated with hypertrophy (with or without dilatation) of the left ventricle, a dilated and atheromatous condition of the ascending portion of the aortic arch is rarely unattended by accentuated second sound in the aorta. Nor is it difficult to explain the connection: the dilated and atheromatized vessel must con-

\* *Edinburgh Medical Journal*, vol. viii., 1862-3.

vey to the surface, more distinctly than in the healthy condition, the sound of valvular tension elicited by the reflected force of an hypertrophied ventricle. In cases of simple or dilated hypertrophy of the left ventricle, with inadequacy of the mitral valve, or with obstruction (with or without inadequacy) of the aortic valve, the inordinate contractile force of the left ventricle is neutralized by the valvular lesion; and similarly, the hypertrophy of chronic renal disease is most frequently rendered incompetent to elicit an exaggerated second sound by the consecutive advance of fatty degeneration of the ventricle. In the early stages, however, of the hypertrophy dependent upon Bright's disease, I have met with examples of intensified second sound; but I cannot recall a single instance in which atheromatous disease of the first portion of the aorta did not likewise exist.

The condition, therefore, in which this sign is most frequently met with, and of which it is in the great majority of cases diagnostic, is the two-fold condition of aortic atheroma and left ventricular hypertrophy; but, inasmuch as the former is usually the antecedent of the latter change, it may be regarded as, *par excellence*, the cause of, and the change indicated by, accentuated second sound in the aorta.

*Transmission of the sounds and impulse of the heart* is influenced by certain conditions of the intra-thoracic viscera which demand notice.

Laennec held that, "1. Tuberculous excavation and pneumothorax transmit the sounds of the heart rather than its impulse, whilst hepatization and compression from effusion occasion results the reverse of these.\* 2. The extent of pulsation (sounds) is in the direct ratio of the thinness and weakness of the heart, and consequently inversely as its thickness and strength. 3. The size of the organ is also favourable to the extent of pulsation, except where increase of size depends on thickness of walls exclusively. 4. The impulse is inversely as the extent of pulsation, and directly as the thickness of the walls of the ventricles. 5. Strong impulse is characteristic of hypertrophy. 6. The absence of impulse is characteristic of dilatation. 7. Hypertrophy with dilatation, the latter being in excess, communicates a

\* *Forbes' Translation*, p. 540.

sensation like the blow of a mallet; it is not extensive, and does not elevate the wall of the chest, and the same result obtains, but in a less degree, in purely nervous palpitation. \*

8. The softening of the substance of the heart renders the sounds much duller than natural." He adds, "In certain healthy subjects, in whom the walls of the heart are thinner than common, the contractions of the auricles are sometimes much louder than those of the ventricles below the clavicles, although the same disproportion is not observed in the cardiac region."

These propositions form a connected and well-ordered series, and I have arranged them in numerical succession for the purpose of more convenient reference.

No. 1 expresses an established and generally-admitted truth, respecting which it is unnecessary to use argument.

There is, however, one species of pleural effusion which possesses in a peculiar degree the property of transmitting the impulse of the heart, namely, empyema. Under certain circumstances, as yet unknown, purulent collections in the pleura exhibit in a high degree the property of vibration, in virtue of which they conduct and even intensify the pulsations of the heart. An example of this kind is given by Dr. Stokes; † the heart was displaced to the right; and the left side, which was the seat of empyema, pulsated throughout so as sensibly to shake the bed on which the patient lay. Yet the force of the heart's action seemed not greater than normal. Two cases of a similar kind, in the practice of Dr. Graves, are reported by Dr. MacDonnell. ‡

The extent to which the sounds of the heart are transmitted, irrespectively of the condition of adjacent organs or parts, is directly as the thinness of its walls, but *not* as the weakness of them. In an advanced state of fatty degeneration of its substance, the heart is often flabby, thin, and weak, yet the first sound is faint and ill-pronounced, and at a short distance from the superficial precordium, not at all audible; the second sound is likewise weak, and not extensively transmitted in the absence

\* *Loco citat.*, substance given, but not exact words.

† *Diseases of the Heart and Aorta*, p. 607.

‡ *Dublin Journal of Medical Science*, vol. xxv., p. i.



of valvular lesion, unless the aorta be at the same time atheromatous, as usually is the case. With this exception the second proposition remains unchallenged.

Increase in the size of the heart, not due exclusively to thickening of its walls, implies absolute or relative attenuation. In the former case increased extent of pulsation will result, under the law expressed in the last proposition, reinforced in this case by the effect of increase in the volume of the heart.

In hypertrophy with dilatation, the condition in which the heart attains the greatest volume, the sounds are heard in front at a distance from the precordium, but they are of a masked or indistinct character; and posteriorly they are similarly diffused, owing to the extensive contact of the heart with the posterior thoracic wall.

The fourth and fifth propositions cannot be questioned, but although the absence of impulse (6th) is "characteristic" of dilatation, it is by no means *pathognomonic* of that condition, as it is no less indicative of softening.

Proposition 7 must be admitted in its entirety, so likewise proposition 8; but the increased loudness of the second sound beneath the clavicles is due, I apprehend, not to thinning of the walls of the heart, but to increased density of those of the aorta, or to resistance in the pulmonary circulation. Again, the subjects of this remark may be simply nervous, and suffering from palpitation; the second sound in the aorta being sharp and ringing, as the result of an abrupt and forcible distention of the aorta, reflected upon the valves through the reaction of its walls.

*The radial pulse*, as the index of the state of the circulation, and remotely that of the nervous system, has been, from the earliest period of the history of medicine, assigned the foremost place in symptomatology. Recent investigations by means of the sphygmograph have invested this sign with importance, scarcely less than it possessed previously to the introduction of percussion and auscultation into practical medicine. By means of the radial pulse we may *approximately* determine the rapidity, force, and rate of contraction of the left ventricle, and by implication, of the other chambers of the heart likewise. Certain forms of disease of the valves and of the substance of the heart,

and certain functional derangements of the nervous system, are also suggested by special characters of the radial pulse, as will be pointed out under the appropriate heads.

The state of tension or relaxation of the arteries, the volume of blood circulating within them, and the degree of resistance of the capillaries, are likewise indicated by the pulse at the wrist. Finally, the state and degree of nervous irritation ; and impaired or suspended activity of the nerve centres, are indirectly represented in the radial pulse.

The rapid contraction of the left ventricle characteristic of the stage of reaction of all continued fevers, and of the hot stage of ague ; of nervous excitement from depressing or exhilarating emotions ; and from sharp but not exhausting pain ; and of uræmic intoxication, is represented by an *abrupt pulsation of the radial artery*. Such is likewise the character of the pulse, but associated with weakness, and often with irregularity or intermittence, where the left ventricle is thin and dilated.

The beat of the radial artery is, on the contrary, *prolonged or drawn out* when the contraction of the left ventricle is sluggish or protracted. This happens especially in dilated hypertrophy of the left ventricle ; it is also witnessed in apoplectic coma. In these conditions the pulse is likewise full, indicating a strong but not vigorous contraction of the ventricle ; in the latter it is tense, but in the former yielding ; a difference due to the vaso-motor irritation of encephalic pressure. The strongest radial pulse is associated with left ventricular hypertrophy and dilatation, the hypertrophy being relatively much in excess ; a condition frequently met with in the advanced stage of organic disease of the kidneys, especially of the cirrhotic form, and before tissue degeneration of the heart has supervened.

The *rate* at which the contraction of the left ventricle is repeated within a given time is ordinarily determined by registering the number of pulsations of the radial artery in the same period. There is, however, one important exception to this rule, namely, in the last stage of many diseases involving partial failure of the heart ; notably in left ventricular dilatation without hypertrophy, in simple softening, and in fatty degeneration of the heart, the number of cardiac pulsations exceeds that of

the radial artery; the difference being due to the occurrence of certain *abortive* contractions of the ventricles, audible, and often even palpable at the precordium, but not represented at the wrist. The systole of the ventricle is blighted or inhibited in its progress, or its walls fail by simple debility to develop a force sufficient to propel the blood to the extremities. Such failure is usually irregular in its recurrence; but in at least one instance I have noticed it to be perfectly regular; an effective contraction of the ventricle, represented by a distinct pulsation at the wrist, being regularly succeeded by an abortive effort, cognizable only by auscultation of the precordium, and totally imperceptible in the radial artery. The patient in whom this remarkable phenomenon was presented was a young and delicate female, convalescent from enteric fever of a mild character, in whom the action of the heart was previously irregular and intermittent. There was no valvular disease, and the sounds were sharper than normal, indicating thinning of the ventricular wall. The alternation of effective and abortive pulsations of the heart continued for a period of twenty-four hours, the action then again became irregular and intermittent. After an unusually protracted convalescence the patient entirely recovered, the action of the heart becoming perfectly regular. This case furnished one of the most notable illustrations with which I have become acquainted, of the specific virtue of digitalis as a *regulator* of the heart's action. After one or two doses of the medicine, containing each five minims of the tincture of digitalis, the action of the heart became regular, and continued so as long as the digitalis was administered; but when it ran out, or was purposely suspended, the action became again irregular and intermittent, as was repeatedly witnessed by the class.

*Infrequent pulsation* of the radial artery, commonly but inappropriately designated "slow pulse," is especially characteristic of certain forms of fatty degeneration of the heart, as shown by Dr. Stokes,\* who regards this phenomenon as indicative of "an equally weakened state of both ventricles."† Mr. Adams was the first, so far as I am aware, to notice the connection between

\* *Dublin Journal of Medical Science*, vol. xi., 1846.

† *Diseases of the Heart and Aorta*, p. 332.

fatty heart and permanently-slow pulse.\* Dr. Henry Kennedy has likewise given special attention to this subject; and, in a memoir published in 1864,† expressed the opinion, that a pulse permanently under 40 is usually associated with the degenerative, as distinguished from the accumulative form of fatty heart.

I believe infrequent pulsation of the heart, in the circumstances mentioned, is directly due to impaired nutritive assimilation or malnutrition of the tissues, especially of the nerve-mass constituting the respiratory centre; a change probably due to arterial degeneration of the parts affected. In such case inhibition of the heart, through the pneumogastric nerves, to the standard of impaired nutritive capacity of the tissues, would seem to follow, under the law of the organism by which functions, mutually dependent, exercise a reciprocally regulative influence. Where vascular atheroma and softening of the respiratory centre had made progress, I should expect, in addition, slow and irregular breathing.‡

*Excessive increase in the rate of arterial pulsation* occurs most frequently in connection with organic disease of the heart; it is usually intermittent and paroxysmal, and associated with palpitation. In one instance I have known the pulse to attain a rate of 210 in a minute. The case was that of a young man (Case 78, Owen Gilson) suffering from mitral and aortic valve disease with considerable hypertrophy of the heart. Ordinarily, the pulse did not exceed 96; but during an accession of palpitation, to which he was subject without any assignable cause, the pulse usually went up to 198, and on one occasion, to 210 in the minute. In accordance with the modern doctrine of pneumogastric inhibition of the heart, this phenomenon might be explained on the assumption that, owing to some unknown cause, the restraining influence of the pneumogastric upon the heart was suspended, and the organ consequently "ran riot."

*The throbbing or collapsing pulse* of permanent patency of the

\* *Dublin Hospital Reports*, vol. iv., 1827.

† *Proceedings of Surgical Society*, March, 1864.

‡ I have not witnessed a pulse-rate under 28 in the minute. Burns (*Observations on Diseases of the Heart*, 1809, p. 46) records a case of fatty and dilated heart, in which, at intervals, and accompanying paroxysms of dyspnoea, the pulse fell to *ten beats* in the minute.



aortic orifice exemplifies pulse of a special character, indicative of a particular form of valvular disease. It is, truly, "the pulse of an unfilled artery" striking the finger feebly and abruptly, and suddenly and completely subsiding. Pulse of this character is always *visible*; but so likewise is the pulse of tortuous and of rigid arteries, corresponding, respectively, to the early and the late stages of atheromatous change of the arteries in middle and advanced age. The devious bending of the artery, its slow distension and gradual subsidence, and the strength and firmness of the pulse in the former condition, serve to distinguish it from the collapsing pulse of aortic patency, which exhibits characters in all respects the reverse; and, in the latter, the rigid state of the vessel will alone suffice for the differential diagnosis.

*Reduplication*, or *dicrotism* of the pulse, is a phenomenon of much interest and diagnostic significance. It is met with under the triple combination of abrupt and feeble contraction of the left ventricle, imperfect distension of the aorta, and diminished capillary resistance. These conditions are supplied in a pre-eminent degree in typhoid or enteric fever, in which, accordingly, after the tenth day, I have rarely missed the phenomenon of dicrotic pulse. It is not, however, peculiar to, or pathognomonic of, typhoid fever. I have noticed it in convalescence from other wasting diseases, especially rheumatism unattended with cardiac complication, and in the anæmic condition associated with lead-poisoning;\* but in an especial manner, and much greater degree, it is associated with enteric fever in the stages of eruption and defervescence.† The pulse is soft and compressible, and the first beat, which represents the normal pulse, and coincides with, or immediately succeeds the systole of the ventricles, is the stronger of the two. The second beat follows‡ by continuous succession; that is, there

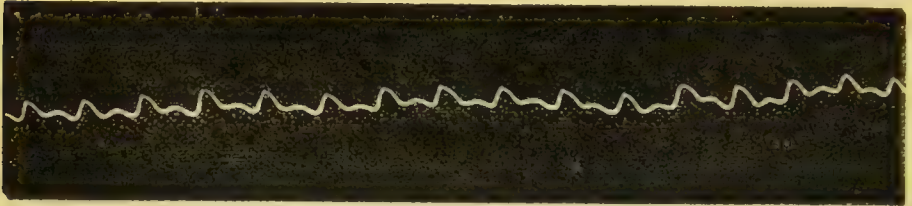
\* *Vide* "Pulmonary Artery," lesions of, case of Hegarty.

† Dr. Waters (*Diseases of the Chest*, second edition, 1873) declares that in pneumonia, he has found the pulse invariably dicrotic.

‡ Czermak has shown, by means of his *photosphygmograph*, that the pulse in the dorsal artery of the foot is 0.018 sec. later than that in the radial artery, and that the latter is later by 0.159 sec., and the pulse in the carotids by 0.087 sec. than the shock of the heart. H. E. Weber estimated the rapidity of the pulse-wave at about 28.5 feet per second; but this must be distinguished from the velocity of the blood itself.

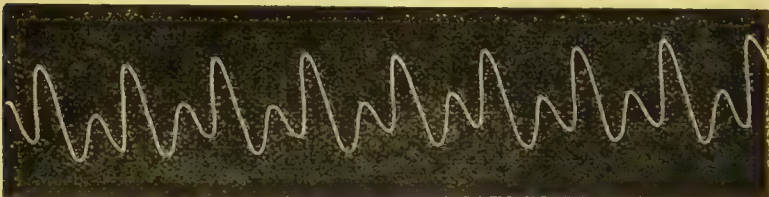
seems to be no distinct interval between the first pulsation of the artery and the second, the stage of retrocession of the former passing, by a gentle curve, into that of ascent of the latter. The subjoined figures (XXII, XXIII, XXIV, XXV, XXVI, XXVII, XXVIII, XXIX, and XXX), well represent the phenomenon of dicrotism under different conditions. The first was obtained from a patient under my own care ; the second and third are borrowed from Lorain.\*

FIG. XXII.



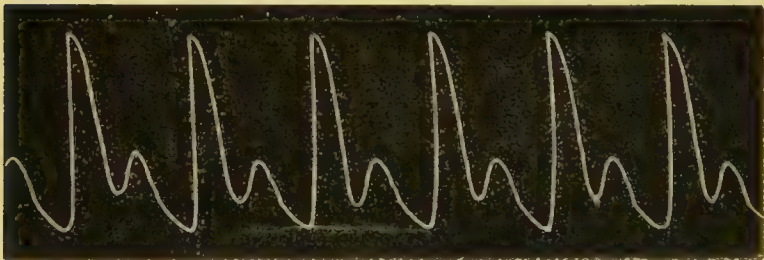
Typhoid (girl, Byrne) 20th day ; dicrotic pulse.

FIG. XXIII.



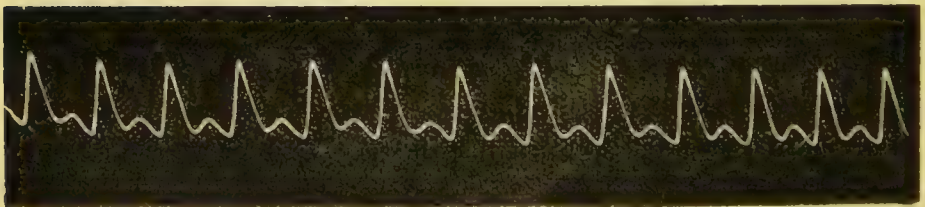
Typhoid fever ; hyperdicrotic pulse.

FIG. XXIV.



Acute rheumatism ; dicrotic pulse.

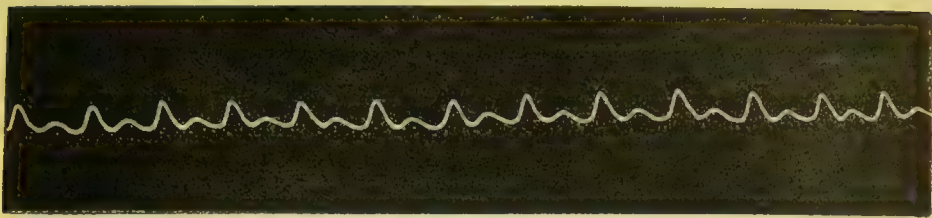
FIG. XXV.



Dicrotism, not perceptible to touch, in a case of severe febricula.

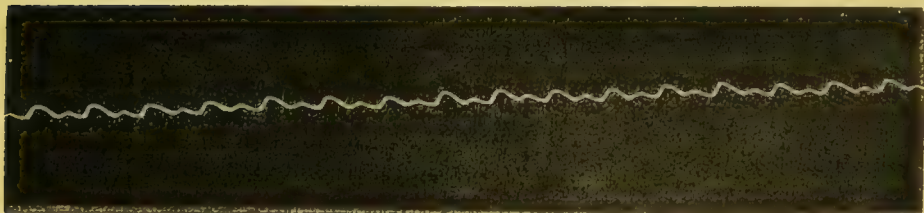
\* *Etudes de Médecine Clinique*, 1870.

FIG. XXVI.



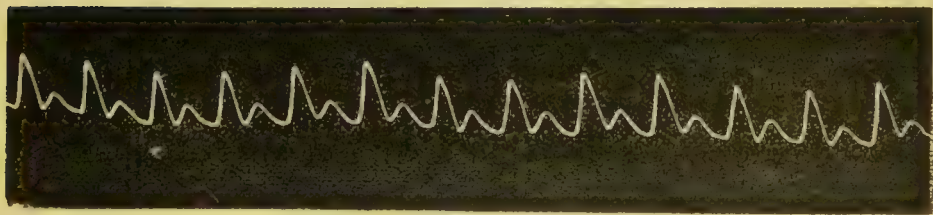
Dicrotism, perceptible to the finger, on the second day of slight febricula.

FIG. XXVII.



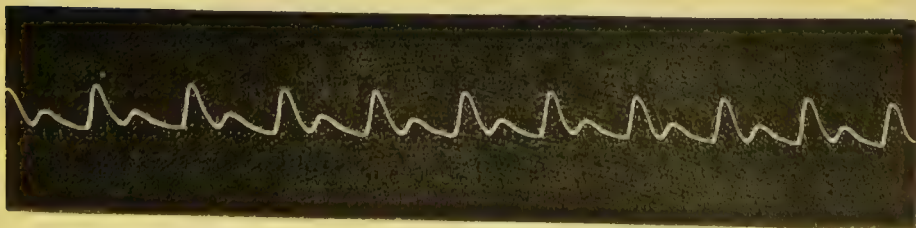
Dicrotism, not distinctly perceptible to touch.

FIG. XXVIII.



Distinct dicrotism, on the tenth day of typhoid fever complicated with pneumonia, and the day following profuse intestinal hæmorrhage. Several of the pulsations are hyperdicrotic.

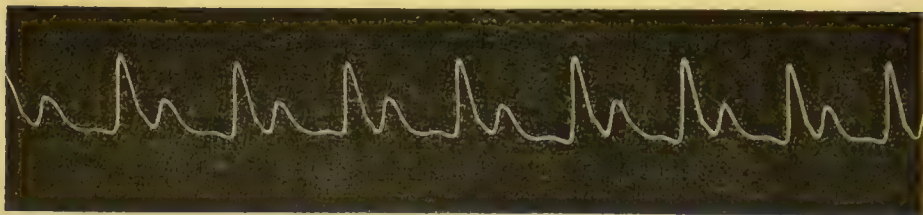
FIG. XXIX.



Dicrotism, on the twelfth day of typhoid fever, quite perceptible to the finger.



FIG. XXX.



Dicrotism, perceptible to the touch, in a case of febrile excitement, accompanied with diarrhoea, after a surfeit of unripe fruit.\*

With few exceptions, authors are agreed in the opinion that multiplication of the arterial pulse, proportionately to that of the heart, is directly due to elasticity in the coats of the arteries. Galen was the first to explain dicrotism by attributing it to vibration of the artery, produced by afflux of blood. Vivenot (de Vienne) says, "The polycrotism is more pronounced in proportion as the artery contains less blood relatively to its calibre, and the more quickly it fills and empties itself." He adds, that elasticity of the artery is likewise indispensable, as exposure to compressed air arrests dicrotism.†

Ludwig regards the second pulse of dicrotism "as a consequence of the elastic action of the first."†

Buisson and Naumann held that the first pulse was due to systole of the left ventricle, and the second to recoil from the sigmoid valves, of blood forced back towards the heart by the systole of the aorta. Naumann satisfied himself, by experiments with his artificial apparatus designed to represent the heart, that dicrotism is incompatible with inadequacy of the aortic valves; for he found that, by partially or completely destroying these, he could reduce or abolish dicrotism.† He likewise found that dicrotism was more pronounced in proportion to the proximity to the heart, of the artery examined, but the undulations more numerous in proportion to its distance from that organ.

In 1864 Landois also experimented with elastic tubes, and confirmed the opinion of Naumann, with whom his results placed him in strict accord.

\* For the last six tracings in this list I am indebted to my friend, Dr. Grimshaw.

† Lorain, *opus citat.*, p. 34-47.



In regard to reduplication, this writer classified pulses under three heads; namely,

1. Those in which there is no reduplication, which he designates as "simple."
2. Those which are repeated in the line of ascent, "anacrotic."
3. Those which are repeated in the line of descent, "katacrotic."

Either of the two latter may be double or triple. Thus :

Anacrotic	{ Anadicrotic.
	{ Anatricrotic.
Katacrotic	{ Katadicrotic.
	{ Katatricrotic.

He held that, ordinarily, in the neck and adjacent regions the pulse was tricrotic.

Wolff found that a pulse incompletely dicrotic became completely so when the body temperature attained 31·8 to 32·4 Réaumur, and "superdicrotic" when a higher temperature was attained. In typhus he has found the pulse superdicrotic at all periods of the day.\*

Marey regards dicrotism as a purely physical phenomenon, dependent upon two conditions; namely, velocity of current, and oscillation of the liquid column alternately from and towards the heart, the result of elasticity of the vessel.† He says "The oscillation which constitutes dicrotism occurs in the arteries of the periphery; this oscillation requires for its occurrence a rapid impulsion of the liquid, and a sufficiently large mass put in motion." He has succeeded in producing it in an elastic tube incompletely filled. The stronger the arterial tension, the greater is the resistance which the left ventricle encounters in propelling the blood into the aorta. If arterial tension falls below a certain degree, the left ventricle contracts with great rapidity, but without corresponding force, and the wave thereby caused travels with extreme velocity. But whilst defective arterial resistance, as shown by Marey, determines a premature arrival of the systolic wave in the peripheral arteries, it must,

\* Lorain (*loco citat.*), to whose exhaustive work I am indebted for the preceding references.

† *Physiologie Médicale, De la Circulation du Sang*, 1863, p. 264, *et suiv.*

by the imperfect tension of the aorta thence resulting, have the opposite effect in regard to the wave of aortic reaction; namely, that of postponing its arrival at the periphery. Hence the asynchronism of the ventricular and the aortic pulse-wave which constitutes dicrotism. Dicrotism is especially common in the typhoid condition, and is of two kinds, which, however, the finger cannot distinguish; viz., dicrotism of ascent, and dicrotism of descent, of the pulsation-wave. The latter, alone, is true dicrotism. He agrees with Bouillaud in holding that the pulse is dicrotic in aortic valve inadequacy; but adds that it is so in the line of ascent only. In such case, dicrotism of ascent is manifestly caused by obstruction at the orifice of the aorta, and dependent upon the roughened, thickened, or tuberculated state of the valves, which, in the majority of instances, is found to accompany regurgitation by inadequacy of the valves. By way of explanation of the phenomena, he says, the jet of blood from the left ventricle passes freely through the large arteries, but is resisted in those of the periphery; from the latter there is a reflux upon the aorta; but the aortic valves being closed, another obstacle is presented to the reflux current at that point, whence the blood "rebounds" in the direction forwards. These oscillations continue till arrested by the succeeding contraction of the left ventricle. He adds, "after the closure of the sigmoid valves there occurs a new centrifugal current which constitutes the secondary or dicrotic pulsation."\* This statement was confirmed by the result of an experiment, consisting in the application of the sphygmograph and Chauveau's registering hæmodromometer to the carotid artery of a horse pulsating dicrotously. The statement and experiment just quoted from Marey, show that he is substantially in agreement with Naumann as to the cause of the second pulsation in dicrotism. The rate of the circulation and the elasticity of the distal arteries exercise an important qualifying influence. Thus, if the blood arrive in the arteries slowly, there will be no dicrotism; and with the same initial velocity, the peripheral arteries being elastic there will be dicrotism, and being inelastic there will not be dicrotism. Thus, in old subjects,

\* *Opus citat.*, p. 274.

owing to the loss of arterial elasticity, there is only imperfect dicrotism; in young persons, on the contrary, it is best pronounced; and in typhoid fever, in which it may be witnessed in the most typical form, the elasticity of the arteries is in excess, because their contractility is in defect. Arterial tension is associated with dicrotism; feeble tension favouring, and strong tension opposing it. Vertical posture is favourable to dicrotism in the arteries of the upper portion of the body, by lessening their tension. So likewise is intermittence of the heart, and for a similar reason, the beat immediately succeeding an intermission being more decidedly dicrotic. In the aorta and carotids of the horse there is always dicrotism, due to closure of the aortic valves; in the carotids of man there is likewise normally feeble dicrotism; but in the radials it is neutralized by the elasticity of the vessels.

The high authority of Marey upon this and all kindred subjects can alone justify the length of the preceding quotations from his work; and, whilst adopting his views in substance, I shall take the liberty of pointing out a few inconsistencies in them.

The alleged reflux of blood upon the aorta, from obstruction encountered in the peripheral arteries, should, of necessity, coincide with the initial period of ventricular diastole, and therefore with the systole of the aorta and the closure of the sigmoid valves; but the systole of the aorta determines a movement of the column of blood contained in it in two opposite directions; namely, towards the heart, by which the valves are shut down; and towards the periphery. The latter must, therefore, encounter and neutralize the alleged reflux from the peripheral arteries, and render it incapable of producing a secondary peripheral pulsation. The typical character of dicrotism in typhoid fever, and under similar conditions in the young subject as contrasted with the aged, I believe to be due, not wholly to a difference in the elastic quality of the arteries as such, but in a great measure to the different degree of resistance presented to the onward movement of the blood, to whatever cause due. I incline to the opinion of Wolff, already quoted, which implies that the dicrotism of typhoid is indirectly

due to the high temperature attained in that form of fever, by which peripheral resistance is lessened, rather than to that of Marey, who, adopting Weber's doctrine of the antagonism and reciprocally inverse manifestation of elasticity and contractility, attributes the dicrotism of typhoid to defect of contractility.

Dr. Burdon Sanderson's opinion on this subject may be collected from the following extract:

"This contraction (of left ventricle) produces expansion of the arteries, and acceleration of the progressive movement of their contents. These two associated and simultaneous effects are not only much more marked in the large arteries than in the small, but occur at an earlier moment, so that at the periphery the current attains its greatest acceleration *somewhat later* than near the heart.

"Hence, at the moment that the ventricle relaxes and the influx of blood through the aortic valve ceases, blood is still moving rapidly onward in the small arteries. As a necessary result, the arterial system becomes relaxed, and the progressive movement of the blood is retarded, collapse beginning where the stoppage occurred (*viz.*, at the aortic valve,) and being propagated towards the periphery. Then, as the capillary arteries are relaxed, the capillary circulation is retarded, while the aorta becomes simultaneously distended in consequence of the increased resistance in front. This distension is, in its turn, propagated towards the periphery, and is succeeded, like the systolic distension, by collapse. If the conditions are favourable, the same series of movements may be several times repeated, the differences between the alternating conditions becoming less and less at each repetition. Thus, we have the following succession of phenomena:

"1. Contraction of the left ventricle.

"2. Distension of the aorta, and greatest acceleration of blood-stream in the great arteries.

"3. Greatest acceleration in the peripheral arteries, occurring simultaneously with cessation of progressive movement in the aorta.

"4. Diminished distension and diminished progressive movement in the aorta.



- "5. Propagation of these effects to the capillary circulation, and consequent increase of arterial resistance.
- "6. Propagation of the resulting aortic distension towards the periphery, producing peripheral acceleration;"\* and so on.

From the preceding extract, although the phraseology is not remarkably clear, it may be inferred that, in the opinion of the author, dicrotism is due to the vibration of elasticity, or alternate distension and reaction of the arteries. The word "collapse" is not strictly applicable to the aorta in the normal state of the circulation; it applies only when the aortic valves are inadequate. In the preceding quotation it obviously means reaction; but as reaction sets in progressively from the aorta towards the periphery, the blood must be propelled in advance of the reaction-wave, and hence there can be no obstruction to the aorta from retardation in the capillaries.

Reaction of the aorta immediately succeeds distension of that vessel, and is simultaneous with the commencement of ventricular diastole. As a consequence of reaction of the arch of the aorta, and proportionate diminution of its calibre, the blood-column which it contains is elongated both in the direction backwards towards the heart, and forwards towards the periphery; the former of these movements is limited and suddenly arrested by the closure of the sigmoid-valves; and, being still under the pressure of the reacting vessel, the retrograde current becomes progressive, in continuation of the primary reaction-wave.† With normal capillary resistance the action and the reaction-wave, or those of ventricular and aortic systole respectively, are in direct and unbroken continuity, and constitute, as

\* *Handbook of the Sphygmograph*, 1867, p. 23.

† I cannot admit the doctrine advocated by Longet (*Traité de Physiologie*, 3ième. edit., 1869; tome ii., p. 172), in the following words. "Immediately after closure of the sigmoid valves, the blood which rests upon them is perfectly immovable till displaced by the next systole." The aorta is elastic, quite to its root, and it is inconceivable that the portion of the vessel adjoining the valves, whilst possessing the same physical properties as the remainder of it, should be quiescent; all other portions being, at the same moment, in a state of energetic reaction. That the blood which rests on the aortic valves is capable of retrograde movement, is proved by the physical phenomena resulting from incompetency of the valves. Why, then, should it be incapable of movement forwards in the direction of the general current?

judged by the tactile sense, but a single expansion of the artery. When, however, the peripheral arteries and capillaries are relaxed, or in a state of defective tonic resistance, the percussion-wave flows off before aortic reaction can take effect; and consequently before the flux of reaction, or tidal wave, setting in from the aorta, can reach any of its branches. Hence double pulsation or dicrotism.

If, in addition to premature escape of the flux of ventricular systole in front, that of arterial systole be incomplete and tardy owing to imperfect distension and feeble reaction of the aorta, the interval between them will be proportionately longer, and dicrotism more distinct in the arteries. Feeble contraction of the left ventricle, by imperfectly distending the aorta, and thereby inducing a weak and slowly-advancing tidal wave, supplies this second condition. Hence it is that in typhoid fever, a disease characterized by feeble circulation and nervous exhaustion, dicrotism is most frequently and most typically presented. I can, to the full extent of my experience, confirm Naumann's statement, that true dicrotism is never associated with permanent patency of the aortic valves; and I therefore incline to the opinion of Naumann and Marey, that a large portion of the reaction-wave of normal circulation and of dicrotism is the product of "recoil" or "rebound" from the closed aortic valves.

Doctor Galabin maintains that the dicrotic wave is not solely due to recoil from the aortic valves; for, as he alleges, "it occurred when they were entirely absent." His experiments were made upon elastic tubes attached to the heart of the sheep, and to an artificial heart of india-rubber.\* I am by no means convinced by Dr. Galabin's experiments. It is notorious that in the living body dicrotism of the pulse is *never* met with where inadequacy of the aortic valves exists.

Dr. F. A. Mahomed adduces† the disappearance of the dicrotic wave of the normal pulse-tracing, which occurs in tracings obtained from the subjects of aneurism of the arch of the aorta, as evidence of the proximal origin of dicrotism,

\* *British Medical Journal*, September 6th, 1873.

† *Medical Times and Gazette*, August 9th, 1873.

rather than of its distal origin as maintained by Dr. Burdon Sanderson.

Niemeyer regards dicrotism as a result of defective tonicity or sub-paralysis of the arterial walls.\* But it should be remembered that, normally, the pulse is slightly dicrotic, although not perceptibly so to the finger.

Dr. Boileau agrees with Dr. Burdon Sanderson in attributing dicrotism to peripheral vascular resistance, although, with Niemeyer, he admits it may be in some degree due to defective arterial tonicity.†

\* *Text Book of Practical Medicine*, 1869, vol. ii., p. 583.

† *Irish Hospital Gazette*, February 16th, 1874.

## CHAPTER III.

### PHYSICAL SIGNS OF DISEASE OF THE HEART.

HAVING now discussed the rhythm of the heart as a concrete phenomenon, and as such it certainly is the most striking manifestation of organic life, whether considered in regard to its regularity, its persistence, or its strict adaptation to the various and important purposes fulfilled by the circulation of the blood; I proceed to consider in this place, as furnishing the most appropriate connexion with which the subject may be introduced, the various derangements to which the rhythm of the heart is liable. These may be considered under five heads; viz., *intermission, irregularity, multiplication, suppression, and inequality* of the pulsations of the heart.

*Intermissions* of the heart have been classified by Laennec\* as *true* and *false*; the former where the action of the heart is really interrupted; and the latter where it occasionally contracts so feebly that the wave does not reach the wrist; hence the radial pulse alone intermits in such cases. Bouillaud† maintains that these intermissions, whether true or false, are regular as regards their period of recurrence. I cannot agree with him in this opinion; on the contrary, intermissions, of whatever kind, are rarely rhythmical in recurrence. In one instance only have I met with rhythmical or periodically-recurring intermissions. A girl aged 22, of nervous and hysterical constitution, and the subject of obstinate abdominal pneumatosis, was received into hospital under my care in April, 1870. She was in apparently good health with the above exceptions; but the cardiac and radial pulse intermitted periodically, at intervals of a few seconds, the sounds of the heart being normal. Under treatment directed to the improvement of her general health, and

\* *Traité de L'Auscultation Mediate*, tome ii., chap. viii.

† *Traité Clinique des Maladies du Cœur*.



especially of the nervous system, the pulse became quite regular, whilst the abdominal inflation continued unabated. I have not met with this symptom precursory to critical diarrhoea, as mentioned by Laennec.

*True intermissions* usually occur at short but unequal intervals of time; they are most frequently met with in old persons otherwise apparently healthy, and associated with a weak cardiac impulse, and sharp clear sounds; the arteries are likewise usually tortuous, and often atheromatous; in short, the symptoms and signs of dilated heart and atheromatous degeneration of the arterial system are usually associated with this form of intermission. It may likewise appear temporarily in convalescence from fever and other protracted and wasting diseases; and finally, in hypertrophy with softening of the heart. The length of the intermission may vary, as stated by Laennec, from one quarter to an entire pulsation of the heart; it usually succeeds the second sound. The existence of intermittence does not, of itself, warrant an unfavourable prognosis. I have known an old gentleman who, from early manhood, had been the subject of intermissions of this kind, and nevertheless, had gone through a laborious professional career, and lived to the age of eighty-one, dying ultimately of bronchitis and asthenia, but without exhibiting any of the ordinary consequences of disease of the heart. This gentleman's sister also died with similar symptoms at the age of seventy-six, and exhibited in her last illness intermitting pulse, with the signs of dilated heart and atheromatous degeneration of the vessels.

*False intermissions* may likewise occur in cases similar to the preceding, but are most frequently met with in connexion with the signs of simple dilatation of the heart, in persons, usually females, of middle age and weak constitution. Intermissions of this kind are occasionally witnessed in conjunction with irregularity, in simple functional derangement, the consequence of vicious habits.

I cannot subscribe the opinion expressed by Dr. Richardson in his otherwise admirable lecture\* on intermittent pulse and

\* "Lectures on Experimental and Practical Medicine," *Medical Times and Gazette*, January 4th, 1868.

palpitation, that in the usual form of intermittency a second sound exists in the absence of a first; and that in the next pulsation succeeding an intermission the second sound is double; nor, assuming the existence of a double second sound as a usual concomitant of intermission, can I admit the explanation given of its occurrence, namely, suspended contraction of the left ventricle alone, whilst the right continues to act, and a consequent disproportion in the number, and asynchronism of the sounds produced by closure of the sigmoid valves on the two sides of the heart.

Separate contraction of one of the two ventricles, the other being contractile but quiescent, is scarcely conceivable, since their nerve-supply, and a considerable portion of their muscular walls, are common to both. No doubt, in the experiments of Schiff already mentioned (page 57), individual portions of the heart were paralysed by deligation of the branches of the coronary artery supplying them with arterial blood, whilst the remainder of the organ continued to act; but in this case contractility was annihilated in the part affected by the experiment, and the heart was thus reduced to the condition of a mutilated organ. In the hypothesis of Dr. Richardson, however, a general or constitutional cause, operating upon the entire nervous system, is supposed to induce *partial* paresis of the cardiac nerves, and paralysis of *one* of the four chambers of the heart. The explanation of reduplicated second sound offered by this author, would imply, moreover, that this phenomenon depends upon, or cannot be dissociated from, intermittence of the heart; but any such assumption is contradicted by the fact that reduplication of the second sound without intermission, is by no means uncommon, as the recorded examples of mitral constriction (see cases) will show. A faint first sound, followed by a proportionately indistinct second sound, as in the so-called false intermissions of Laennec, is of frequent occurrence, and quite intelligible; but a second sound, not preceded by ventricular systole, is to me incomprehensible. On the other hand, I have, though rarely, met with examples of intermission supervening immediately on the first, with loss of the second sound.

There may be *apparent* intermissions where two beats of the heart occur in rapid succession, followed by a prolonged pause, the prolongation, however, being in the direction *backwards*; that is to say, owing to the anticipation of the normal period of its occurrence by the preceding pulsation, the subsequent pause is proportionately prolonged. The apparent intermission is, therefore, no other than the normal long pause, with a prefix due to the premature occurrence of the preceding ventricular systole.

*Irregularity* is of much more frequent occurrence than intermission, though occasionally, and in certain cases, such as those of functional derangement, often associated with it.

Irregularity is, *par excellence*, the sign of cardiac neurosis; and I quite agree with Dr. Stokes\* in the opinion that, where associated with organic change, "it is more intimately connected with lesion of the muscles than of the valves of the heart." Hope† justly regards irregularity as of more serious import than intermission of the heart. This is, no doubt, due to the fact that it is more frequently associated with organic lesion; and when it occurs in connection with valvular disease, there is invariably present likewise tissue change of the heart-substance, to which, rather than the valvular lesion, the irregularity owes its origin. In cases of acute disease of the valves, even though disorganization of their structure be already complete, irregularity is never witnessed unless when directly traceable to some other cause. Myocarditis, and pericarditis with serous effusion, usually give rise to irregular action of the heart. This symptom has been likewise observed by Graves as a precursor of pericarditis. It may occur in a transient form in connexion with gout and dyspepsia, and in an aggravated form, though amenable to sustaining and stimulant treatment, in what might be designated acute failure of nutrition in a weak and dilated heart. In all the foregoing examples of irregularity there is likewise inequality of the pulse; that is to say, the beats are unequal not only in frequency, but likewise in force;‡ and there is no

\* *Diseases of the Heart and Aorta*, 1854, p. 175.

† *On the Diseases of the Heart*, 3rd edition, 1839, p. 377.

‡ "*Faux-pas*" of Bouillaud.

standard proportion observed as between them in either of these respects. The irregularity is, therefore, *arythmical*.

There is, however, another form of irregularity in which the rate varies within a period of a few seconds; but the beats both of the heart and pulse are strictly proportionate to one another as to frequency and force, under each of these rates respectively. This form of irregularity, which may be distinguished from the preceding as *rhythmical*, is strictly functional, and due to nervous derangement; it is of favourable augury, and according to my experience, exemplified only in cases of nervous debility consequent on masturbation, or the immoderate use of tobacco or of tea.

Dr. Richardson points out two forms in which irregularity of this kind, consisting in rapid changes in the rate of the pulse, may be exhibited. One he names "acute irregularity," in which the pulse beats at different rates in a series of five to ten or more pulsations; the members of the different series being strictly proportionate to one another in time, but differing in this respect from those of other series. This condition of pulse he has met with in cases of very feeble heart, in chronic anæmia, and after loss of blood or other serious depressing influence. The other form he names "prolonged irregularity," differing from the former only in this, that the series consist of a greater number of pulsations, and that it is best exemplified in the acute hydrocephalus of children, of which it is almost pathognomonic and of the worst augury.

I have never met with rhythmical irregularity of either kind under the circumstances mentioned by Dr. Richardson; indeed, in the conditions specified by him, I would rather expect to find examples of the first-mentioned, or arythmical form of derangement.

*Inequality* of the pulsations of the heart is always met with in association with false or apparent intermittence, of which, indeed, it constitutes an essential feature; and frequently likewise with arythmical irregularity, so often associated with the preceding derangements. Inequality in the action of the heart of necessity involves a similar anomaly of the pulse; and a case, by no means uncommon, in which all the foregoing derange-



ments are exemplified,\* with the addition of a murmur, or perhaps more than one such, presents a jumble of sounds and movements of an apparently inextricable character, to be correctly interpreted only by those who, by patient study, have acquired a ready and precise knowledge of the normal rhythm of the heart.

*Palpitation* consists in rapid and tumultuous action of the heart, with or without derangement of rhythm, and painfully sensible to the patient. It comes on in paroxysms, during which the large arteries throb, and the nervous system is in a state of great excitement and tension. The attack is usually brought on by mental worry, bodily fatigue, dyspepsia associated with flatulence, or by late and heavy suppers ; but where organic disease of the heart exists it may occur irrespectively of any of these causes. Palpitation may be the disease ; that is to say, no organic lesion, so far as can be made out, co-existing with it. In such cases, which constitute a large majority of those in which it is met with, palpitation may be regarded as a veritable neurosis ; and the diagnosis rests mainly upon the negative evidence afforded by a physical examination of the heart, but in some measure likewise upon observation of the case, and the result of treatment. Thus, for example, it will be noticed that exercise in the open air and cheerful society produce a markedly beneficial influence upon the patient, warding off the attack which generally occurs at night, and insuring sound sleep and improved digestion. In many cases it is due to latent gout, and is readily cured by colchicum. The case of Mr. R.,† is a good example of palpitation due to this cause.

Palpitation may complicate organic disease of the heart, although it is present only in a small percentage of such cases. Dilatation of the left ventricle, with or without hypertrophy, and irrespectively of valvular lesion, constitutes the condition in which, *par excellence*, it is an associated symptom of organic disease. Attacks of this kind are much more protracted and obstinate than the preceding, the most efficacious treatment in the attack being antispasmodics, especially Hoffman's anodyne with

\* *Folie des Battements du Cœur* (Bouillaud).

† *Vide* "Neuroses."

opium; and, in the intervals between the attacks, quinine, strychnine, and iron. Case 68 appropriately illustrates these remarks.

One or other of the two sounds of the heart is not unfrequently found to be *reduplicated*. This occurs much more frequently in regard to the second than the first sound; although Hope, whilst admitting reduplication of the latter,\* which he attributes to impulse of the apex out of time with the first sound, in which, according to him, it normally has no share, makes no allusion whatever to doubling of the second sound. Doctor Stokes† gives several examples of the latter, which he justly regards as the more frequent, and whilst confessing his inability to explain the phenomenon of reduplication, expresses the opinion that it is due rather to valvular than to muscular action, and is probably the result of a want of synchronism in the action of the two sides of the heart.

In regard to relative frequency of occurrence, reduplication of the second sound, according to my experience, greatly preponderates; of this several examples are given in the sequel, the great majority of which have occurred in connexion with obstruction at the mitral orifice.

Geigel and Guttemann state‡ that reduplication of the second sound has been pretty generally met with in connexion with mitral narrowing or inadequacy. Most of the patients were thin and anæmic, and chronic sufferers from heart-disease. The reduplication may be heard over the tricuspid, aortic, or pulmonary orifice, but is most distinct in the two latter situations; and at the site of the pulmonary opening the second of the two elements is louder than it is over the aorta, or is accentuated, in mitral valve-disease. They account for the phenomena by assuming derangement of synchronism in the closure of the two sets of sigmoid valves; the right ventricle being engorged in mitral disease, empties itself more slowly than the left; hence, say they, closure of the pulmonary sigmoid valve is posterior in time to closure of that of the aorta.

They regard doubling of the second sound, when *constant*, as diagnostic of stenosis or inadequacy at the mitral orifice. It may

\* *A Treatise on the Diseases of the Heart*, 3rd edition, p. 42.

† *Diseases of the Heart and Aorta*, p. 118.

‡ *Arch. Général de Médecine*, June, 1869.

exist also in stasis or engorgement of the right side of the heart, to whatever cause due; in cases of dilated pulmonary artery; in young tuberculous subjects; in the subjects of pulmonary emphysema, pleurisy, and fatty disease of the heart; but in these several cases they assert that it would not be constant as in mitral valve disease.

Guttemann does not agree with Geigel in the opinion that doubling of the second sound is an absolutely constant sign of mitral constriction, and present only when the patient is in a state of complete repose; he admits that when the heart's action is quickened the reduplication ceases. I agree with M. Guttemann in the opinion that reduplication of the second sound is by no means constant in mitral constriction. It has occurred in only twenty-six out of sixty-three cases in my tables.

Mr. Arch. Bleloch\* suggests that reduplication of the sounds of the heart may be due to interruption, in the cardiac plexus, of the wonted communication between the nerve-currents destined for the supply of the two sides of the heart. The simultaneousness of influx of the *vis nervosa* into the two sides of the heart would be thus prevented, and synchronous contraction consequently precluded.

He also offers, as another explanation, that whereas in man the *vis nervosa* travels at the rate of two hundred feet in a second, and in the frog only at the rate of ninety feet, neurosis of the heart may consist in "retrogression for the time being to a lower type of nerve-tissue;" *e.g.*, that of the frog. If, in consequence of unilateral neurosis, one side of the heart should undergo thus a slowing of its rate of action, "both the rate of transmission and the quantity of nerve-force transmitted may be less," and so derangement of synchronism of action take place. The first hypothesis is entirely gratuitous, being unsupported by evidence of any kind; and besides, it would not apply to examples of *temporary* reduplication. The second hypothesis, though exceedingly ingenious, is not only unproven, but in the present state of knowledge, not susceptible of proof or refutation.

Of reduplication of the first sound I have noted in all twelve cases; viz., eight females and four males. Of these six were

\* *Medical Press and Circular*, June 22nd, 1870.

set down as "nervous," three being simply so with menstrual derangement; and of the three remaining, one (a male) was gouty, one (a female) was the subject of Graves' disease (exophthalmic goitre), and the third was an example of mitral constriction, both sounds being double. Hypertrophy of the left ventricle existed in three cases; viz., in two with mitral reflux, and therefore with some degree of dilatation also; the remaining example being one of simple hypertrophy consecutive to cirrhosis of the kidneys. Dilatation of the heart in connexion with cirrhosis of the liver existed in one case, and in two there was fatty degeneration of the heart.

I have met with reduplication of *both* sounds in two patients; one, above referred to, was a female aged thirty years, and the subject of contracted mitral orifice; and the other, a female aged seventy-five, suffering from bronchitis. In this latter case three sounds were audible at the base and at the apex respectively; but in these two situations the doubled sound was not the same.

The causation of this phenomenon must be considered separately in regard to each of the sounds.

When associated with the first sound, I am satisfied it is due to resolution of that sound into its two normal elements, namely, the cardiac impulse, and the sudden tension of the auriculo-ventricular valves. In most of the cases it was possible to identify with great facility these two elements, the dull thud of the impulse having been, in every instance, antecedent to the sharp click of valvular tension. Cardiac impulse coincides with the initial portion of ventricular systole, whilst tension of the auriculo-ventricular valves and attached chordæ tendineæ occurs at the conclusion or acme of systole. Under ordinary circumstances the act is so rapid in accomplishment, that the two elements which enter into the first sound by which it is announced, are virtually simultaneous in occurrence. Not so, however, when any cause intervenes to protract the systole. In that case the initial portion of the first sound is dull, because due to the stroke of the apex only; whilst the concluding portion, if the valves be in a condition to yield, the sound of tension on being suddenly put upon the stretch, is sharp and clear, because no longer masked by the dull element of impulse. In the event, however,



of the existence of valvular lesion competent to neutralize the sound of valve-tension, or to mask it by a coincident murmur, the impulse-element alone will represent the first sound, which, in such case, is necessarily of a dull or masked character, whilst a murmur, *post systolic* in time, represents the second element.

The organic lesions with which reduplicated first sound is associated are; in point of fact, of the above-mentioned character. Thus, of twelve cases in which this anomaly has been exhibited in my experience, four were examples of hypertrophy with dilatation of the left ventricle, and two of fatty heart. The condition of heart, however, with which, *par excellence*, reduplicated first sound is associated, is that of nervous instability, as exhibited in proneness to palpitation. Of this, no less than six of my cases are examples; but of these, one was also gouty, and one exhibited contraction of the mitral orifice and reduplication of both sounds.\*

*Doubling of the second sound* is a phenomenon of a more distinctive character, and more frequent occurrence than the preceding. It is more easily appreciated because of the clear and decisive character of both its elements. It is due to derangement of synchronism in the closure of the two sets of semilunar valves, as the following observation, which I have repeatedly made, seems to prove. In every example of this anomaly which has come under my notice, the double character of the sound was exhibited only in the area over which the sounds of the aorta and the pulmonary artery were *both* audible; whereas, when the stethoscope was shifted a short distance to the right or left of this region, a single second sound only was heard.

Of twenty-seven examples which I have noted of reduplicated second sound, no less than twenty-six were cases of constricted mitral orifice, the remaining one being an example of aortic obstruction and reflux.†

\* Reduplication of the first sound is of very frequent occurrence in simple hypertrophy of the left ventricle. In the cases above mentioned it would seem to be due to *postponement* of tension-click, and in simple hypertrophy to *præponement* of impulse.

† Between the dates at which the paragraph on this subject at p. 129 and the above were written, I had met with the additional examples of this phenomenon included in the number now given. Since the latter date I have noted several others, some of which will be found amongst the reported cases *passim*.

In the former cases it is not difficult to understand how, owing to imperfect distension of the left ventricle in diastole, and consequently of the aorta during ventricular systole, the reaction of this vessel, and the closure of its valves, shall occur prematurely, and be anterior in time to those of the pulmonary artery, and a double second sound be accordingly developed. The engorged state of the pulmonary artery consequent upon the obstruction in front, will contribute to the same result, by postponing the reaction of that vessel and the closure of its valves.

Where reduplication of the second sound is associated with valvular inadequacy at the orifice of the aorta, of even a few month's duration, the left ventricle may be assumed to be dilated. In such case the evacuation of that chamber is of necessity protracted, and in a still greater degree where obstruction at the mouth of the aorta coincides with permanent patency of the valves. Hence, the reaction of the aorta is delayed, and the aortic element of the second sound is dissociated from that of the pulmonary artery by postponement. Ultimately, however, owing to tissue-degeneration of its walls, the dilated left ventricle will fail to evacuate itself, whilst the right ventricle and the pulmonary system have become engorged. From this will follow the twofold result of an earlier reaction of the aorta, and a later reaction of the pulmonary artery. Unification of the second sound is the consequence of this pathological adjustment, and a double sound no longer exists. Such a result is obviously not to be expected, and in fact is never witnessed, in connexion with mitral stenosis.

Where mitral obstruction and permanent patency of the aortic valves co-exist, reduplication of the second sound will not occur, at least in the advanced stage of the twofold disease, when dilatation of both ventricles has ensued.

In Dr. Fuller's excellent work\* there are some passages on this subject which I cannot subscribe. He says: "In all cases its [reduplication of sounds] essential cause is absence of synchronous action in the valves of either, or of both sides of the heart. Thus, if one ventricle contracts before the other, the auriculo-

\* *Diseases of the Chest*, p. 488.

ventricular valves on the two sides of the heart will not close simultaneously, and the first sound will be reduplicated; if the elasticity and irritability of the pulmonary artery and the aorta be unequal, the one vessel will probably contract before the other, and thus cause a want of synchronism in the closure of the semilunar valves on the two sides of the heart.

“But in some instances the reduplication appears to be confined to one side of the heart.

“In these cases there is *a want of synchronous action between the different segments of each set of valves*;\* of the mitral or of the tricuspid, as the case may be, in the first instance; and of the aortic or of the pulmonary semilunar valves in the second instance.”

In the preceding passages the valve-theory of the production of both sounds is exclusively adopted, and all other causes as contributory are excluded. This is not only not in accordance with, but directly contrary to, the bulk of evidence, experimental and clinical, on the point in question. As to the isolated contraction of either ventricle, upon which Dr. Fuller's theory rests, I have already given reason for doubting the possibility of its occurrence.

The aorta and the pulmonary artery are simply passive in regard to the circulation of the blood, and the time and force of their reaction depend not absolutely upon their elasticity, but upon the length and energy of the preceding contraction of the respective ventricles. The contractility of these vessels is, therefore, a borrowed property, and represents that of the corresponding ventricles.

As to the alleged want of synchronism in the closure of the different segments of each set of valves, adduced to account for the phenomenon of reduplication on *one* side of the heart, it is simply a speculation without basis or warrant of any kind, and opposed to everything that is known as to the function of the valves. Manifestly, the individual segments of any of the valves of the heart cannot be closed out of time with the others, and with a force sufficient to elicit from them a sound of tension, without being accompanied by a murmur of reflux; but it is

\* The italics are not in the original.

notorious that a murmur of any kind is a very rare accompaniment of a reduplicated sound at the same orifice.

Dr. Milner Fothergill regards the reduplication of the second sound as *prima facie* evidence of pulmonary engorgement, whether cardiac or not.\*

But reduplication of the second sound is not even an exceptional symptom of simple pulmonary engorgement.

There must be, therefore, some other cause in operation where reduplication exists; and the association, which is undoubtedly frequent, of pulmonary congestion and double second sound, is the compound result of that common cause. (See p. 127.)

The phenomenon of *suppression* of either sound of the heart is strictly and exclusively morbid. *The first sound* is masked in hypertrophy, and all but extinguished in advanced fatty degeneration of the heart; and in typhus-softening, as pointed out by Dr. Stokes,† it is absolutely and entirely suppressed. The phenomenon is easily accounted for by reference to the morbid alteration in the muscular structure of the heart, in consequence of which, although sufficient to maintain a circulation adequate to the wants of the lowest degree of vitality, and barely to avert death, it is incapable of developing the energy required to produce impulse or systolic sound. In syncope, and as a precursor of death, failure of the first sound is a well-known symptom; and this sound may be masked or entirely superseded by a murmur.

*Suppression of the second sound* is of much less frequent occurrence than that of the first. As far as I know it is met with only in the collapse stage of cholera.‡ It is probably due to extreme cardiac debility; but why, with an audible first sound, it should cease to exist in this disease, I cannot satisfactorily explain. Nor am I satisfied with the explanation of Dr. George Johnson,§ who accounts for it on his favourite hypothesis of arrested pulmonary circulation; for although in such case the left ventricle, being deprived of blood, would be incapacitated for the production of a second sound, the right ventricle, for the opposite

\* *Lancet*, November 6th, 1869.

† *Dublin Medical Journal*, vol. xv., 1839, p. 1.

‡ See *Report of Cholera Epidemic of 1866*, by Drs. Hayden and Cruise.

§ *British Medical Journal*, 1868.



reason, should be capable of producing one. Yet nowhere in such cases is a second sound to be heard. Both sounds are masked for obvious reasons in hydropericardium, and in pulmonary emphysema as usually met with, namely, that engaging the anterior edges of the lungs. A condition the opposite of that just discussed, namely, that of exaggeration of the sounds of the heart, is of rather more frequent occurrence.

*Exaggerated first sound* is met with most frequently in connexion with dilated left ventricle; it is sharp, clear, and of brief duration, giving the impression of having originated in close proximity with the ear, and of having been transmitted through a thin and vibratile medium. In extreme ventricular dilatation, when the heart has been excited to vigorous action by bodily exercise or mental disturbance, the quality of the two sounds is so nearly similar that it is exceedingly difficult to identify them individually. But in dilatation and thinning of the left ventricle associated with tissue-softening, the first sound is abrupt, feeble, and usually ill-pronounced, and the action of the heart and pulse rapid and irregular, as pointed out by Dr. Stokes;\* and finally, in typhus fever similar phenomena are exhibited, as mentioned by the same authority,† and both sounds not unfrequently assume a foetal character, becoming weak and almost identical, whilst the action of the heart is exceedingly rapid. In the rare form of disease of the heart described by Dr. Ormerod‡ under the name of “universal fibrous transformation,” the sounds of the heart are likewise of the foetal character, and individually difficult of identification.

*Exaggeration of the second sound* is of more frequent occurrence in the pulmonary artery than in the aorta. In the former of these vessels its occurrence is, according to Skoda,§ pathognomonic of mitral obstruction.||

My experience does not tend to confirm this statement. I

\* *Dublin Medical Journal*, vol. xxi, 1842; *Pathological Society's Report*, p. 133.

† *Ib.*, vol. xv.

‡ *British Medical Journal*, August, 6th 1864.

§ *On Auscultation*, Markham's translation.

|| Dr. W. Begbie (*opus citat.*) regards accentuated, or “booming” second sound in the aorta as suggestive of atheroma of the arch of the aorta, or of aneurism of its transverse portion.

have met with accentuated or intensified second sound in the pulmonary artery in only fifteen out of sixty-three cases, or less than one-fourth of the whole number of examples of narrowing or obstruction of the mitral opening; but in a large proportion of such cases, viz., twenty-six in sixty-three, as already stated, I have observed it reduplicated.

The extent to which the sounds of the heart, being normal, may be transmitted through the chest, depends upon many conditions having reference chiefly to the age of the subject, the configuration and conducting properties of the thoracic parietes, and the state of contiguous organs.

In infants and children, owing to the vibratile structure of the chest at this age, the sounds are transmitted to a greater distance, and with much greater clearness than in the subsequent periods of life. In those with narrow chests, especially of the pigeon-conformation, the sounds are likewise better transmitted, because of the absolute and persistent contact of the heart with the anterior thoracic wall.

Compressed, hepatized, or otherwise solidified lung, serves as a better conducting medium for the sounds of the heart than healthy lung substance.

Laennec asserts\* that the sounds of the heart are better conducted than its impulse through tubercular vomicæ, and through the air of pneumothorax, whilst the reverse is the case in regard to compressed or hepatized lung.

Bouillaud holds that sound is transmitted through the latter no less distinctly than impulse. In this, I think, he is wrong. I apprehend that in all these cases alike the accompanying solidification is the cause of the exaggerated transmission, not only of the impulse, but of the sounds also. The walls of a tubercular cavity, being solid, and in immediate contact with air more or less stationary, are in the most favourable condition for transmitting sonorous and motor vibrations; and that motion should be better conducted than sound through a solid body, including in its interstices much liquid, such as a hepatized or compressed lung, will be readily understood. The pulsatile pneumonia of Dr. Graves affords a good illustration of this

\* *The Diseases of the Chest*, Forbes' translation, 1827, p. 540, foot note.

remark.\* In this singular affection the throbbing of the chest must be regarded as an epi-phenomenon, dependent, according to Graves, upon the engorged state of the pulmonary vessels, which are thereby constituted good conductors of the cardiac pulsation.

Morbid alteration in the quality of the sounds of the heart, constituting *murmur*, has been, since the introduction of auscultation by Laennec, the subject of ardent and no less profitable study. It is essentially a morbid phenomenon, and therefore appertains exclusively to the domain of pathology, but is not remote from the boundary line between health and disease.

A murmur may be *defined* as an abnormal sound developed within the heart, the blood-vessels, or an aneurism; in the heart associated with, or superseding the normal sounds; in the arteries, in aneurisms, and in certain rare cases in the veins, observing the rhythm of the cardiac sounds; and in veins usually continuous, but influenced as to intensity by the sounds of the heart.

*Causes of murmurs.* Before the time of Laennec murmurs were not recognized as morbid sounds; and by him they were attributed to "spasmodic contraction of the heart or arteries."† This opinion seems to have been based mainly upon the fancied resemblance which the typical murmur (*bruit de soufflet*) bore to the sound yielded by muscles during contraction.

Corrigan,‡ as the result of a number of experiments on living cold-blooded animals, and others ingeniously contrived with a view to determine the modifications of sound in membranous conduits under various conditions, arrived at the conclusion that murmur is mainly due to a "current-like motion" of the blood within the heart or vessels. Thus, according to his doctrine, when the auriculo-ventricular orifice of either side of the heart, or an artery, has suffered absolute narrowing by organic disease, or by pressure; or relative constriction as the result of simple dilatation of either ventricle, or of aneurism; the blood in transit through the contracted passage, or into the dilated ventricle or sac, is thrown into a state of intrinsic commotion, or "currents,"

\* *Clinical Lectures on the Practice of Medicine*, 1864, p. 472.

† Forbes' translation, p. 559.

‡ *Lancet*, April 4th and 11th, 1829.

whence arises murmur. The transmission of murmur so originating, as well as variations in its intensity, depends mainly upon the degree of tension of the walls in contact with the moving liquid. In the heart unaffected by disease also, a faint murmur must be developed at the auriculo-ventricular orifices, owing to the difference in diameter between these openings and the corresponding ventricles in complete dilatation; "accordingly in the healthiest there is some sound always accompanying its action;" and in dilatation of the ventricles to any extent the sound is loud, and the degree of sound bears a proportion to the size of the ventricle, or to the degree in which this current-like motion exists. In simple dilatation there is a loud, soft, diffused murmuring sound, from which *bruit de soufflet* differs only by its sharpness. He describes the several steps of an experiment, consisting in the transmission of a strong and rapid current of water through a piece of intestine, performed with a view to ascertain precisely the conditions under which *bruit de soufflet* was developed, and its immediate cause; and adds, "whilst the intestine was tense no sound, or a murmur exceedingly indistinct, was heard; but any part being constricted so as to produce an alteration in the motion of the fluid, a very loud *bruit de soufflet* immediately became evident;" the *bruit* was audible, however, only on the distal side of the constriction.

There can be no doubt whatever that the intrinsic collision of the moving liquid, to which alone Sir Dominic Corrigan attributes the occurrence of *bruit*, is a potent, but not the sole cause of this phenomenon. I cannot admit the exclusion of other and no less effective causes, namely, friction and parietal vibration. Moreover, a murmur is actually heard under all circumstances in connexion with liquids moving with a certain degree of rapidity and force through conduits, whether organic or metallic, and quite irrespectively of the tension of their walls. In the leaden pipes laid for the new water-supply of the houses of Dublin from the river Vartry, the water, moving under a pressure of 50 lbs. to the square inch, yields a roaring noise audible at a distance of several yards, and actually stunning to the ear brought into close proximity with it; and in a hose composed of india-rubber, and connected with the water-



pipe for use in my stable, when in action, a murmur is likewise audible, but much less loud and harsh than the former, and rather of a blowing character; differences no doubt due to the better conducting qualities of metal as compared with caoutchouc.

I cannot admit that in a heart the subject of simple dilatation of either ventricle, or of both ventricles, without other organic change, such as narrowing or roughening of the orifice, an afflux-murmur may occur at the auriculo-ventricular opening; and I entirely deny that in a heart free from disease of any kind, a murmur or sound, however faint, is associated with the transit of blood from auricle into ventricle. Such a murmur should be audible at the apex, either coincidently with or immediately succeeding the second sound, when the influx of accumulation takes place; or immediately antecedent to the first sound, when the auricles contract with vigour. It would be, therefore, either a veritable apex-diastolic or post-diastolic, or a presystolic murmur. Of all cardiac murmurs an apex-diastolic is the rarest, that is, a murmur audible at the precordium within the area of apex pulsation only, and strictly coinciding in time with the second cardiac sound. Out of several hundred cases of cardiac disease carefully examined and noted, I can only recall two examples of murmur answering to this description.

Presystolic murmur, until very recently incorrectly designated diastolic,\* is *sui generis*, and therefore unmistakeable. This murmur, the special significance of which will be discussed at length further on, is now pretty generally known, and can occur only in connexion with narrowing of either auriculo-ventricular orifice.

Hope† states that "valve-murmurs are occasioned by collision of the particles of the blood against each other, and against the containing solids," and subsequently more at length.‡ "The fact, then, respecting murmurs briefly is, that they may be produced by the vibrations either of the liquid alone, or of the liquid and solids conjointly; and the latter is, without doubt, the more

\* *Diseases of the Heart*, by Dr. Hope, 3rd edition, 1839, p. 78.

† *Opus citat.*, p. 80.

‡ *Opus citat.*, p. 81, foot note.

frequent case in the heart and arteries, because these solids are elastic. Hence it is that vibratory tremour is in many instances perceptible to the touch." According to Hope, then, murmur is the result of vibration, whether of the containing parietes, or the contained liquid, or of both; and inferentially, whilst vibration may originate in, and be confined to, the blood, it is, when present in the adjacent solids, always derived from the blood by communication or by friction. Herein, no doubt, lies the truth in regard to this question.

Inorganic murmurs are, according to Hope, attributable to a threefold cause, viz., attenuation of blood, unfilled arteries, and velocity of current; the first and last mentioned causes giving rise to increased friction, the first and second favouring the occurrence of vibration in the blood, the second supplying the condition for the communication of it to the walls of the vessel, and the third being the immediate active cause by which friction and vibration are developed, and in proportion to its degree, intensified. Non-organic murmurs, therefore, equally with organic, are the result of vibration and friction.

In experiments on dogs performed by Hope and Marshall Hall, repeated and copious bleedings caused rough systolic cardiac murmur, sharp and strong impulse, and arterial thrill and throbbing. These phenomena disappeared under two opposite sets of conditions, viz., when the animal was greatly reduced by loss of blood, entailing loss of contractility in the heart; and when the blood was renovated, and thus, at the same time, its volume was increased, and its solid elements proportionately augmented.

When from weakness, murmur, thrill, and throbbing were arrested, they were restored by suspending the animal by the fore legs, manifestly because, owing to the imperfect circulation in the brain in this unnatural posture, the heart and arteries became partially filled, and so, at the same time, the stimulus of imperfect distension was given to the heart, and the conditions for vibration of the blood and of the vessels were again furnished.

Marshall Hall held that vibration was modified, and this murmur caused, by reduction in the quantity or pressure of the

blood, change in the proportion of its elements, and probably in its velocity.\*

Gendrin† holds that murmurs are caused by alteration of structure of the heart or arteries, and changes in the constitution of the blood; and that in the latter case murmur may be heard in all the arteries of the body of a certain calibre.

So-called blood-murmurs are, no doubt, in aggravated cases, audible in the larger arteries of the body generally, but this admission does not imply that such murmurs are regarded as due exclusively to an alteration in the physical constitution of the blood. On the contrary, I believe, and shall endeavour to show in the proper place, that murmurs of this kind are due to nervous reaction upon the walls of the vessels, in no less a degree than to modification in the quantity or in the quality of the blood.

Bouillaud‡ considers valvular murmur to be nothing more than exaggeration, absolute or relative, of the normal bruit arising from the friction of the blood against the edges of the orifice in its passage through, which is a contributory cause of the first sound of the heart; the other and principal cause of that sound being, according to him, sudden tension of the valves, as previously stated at length (p. 95). If from any cause the force of friction be increased, whether from narrowing of the orifice, or from increased force of ventricular contraction, the friction-element of the first sound is thereby *absolutely* intensified, and becomes murmur.

If, on the other hand, the valve-element of the first sound be partially or entirely suppressed by organic change, then the friction-element is *relatively* exaggerated, or brought prominently forward, and so becomes the cause of murmur. Where the valves are rendered, by organic alteration, incapable of moving, a murmur *replaces* the first sound; but in cases in which the valves move at all, although less freely than in the state of health, the normal sound persists; if there be, however, at the same time, narrowing of the orifice, or a rugged or inadequate state of the valves, *bruit de soufflet*, *bruit de râpe*, or *bruit de scie*

\* Gendrin, *Leçons sur les Maladies du Cœur*, pp. 14 and 15.

† *Opus citat.*

‡ *Traité Clinique des Maladies du Cœur*, 1835, tom. i.

will *accompany* the first sound. He holds, therefore, that when the valve-element of the first sound is entirely suppressed by structural disorganization, one of the above-mentioned causes of murmur being at the same time present, a murmur of substitution is the result; but that when any of these causes co-exist with valvular mobility, even though the valves be rough and inadequate to close the orifice, a murmur of accompaniment or a murmur *with* a first sound is heard.

It will be perceived, on reference to Chapters I. and II. (pp. 96 and 115,) in which the sounds of the heart are discussed, that the mere movement of the valves is an element of only secondary value in the causation of them; sound depending upon the structural integrity or healthy condition of the valves, in virtue of which they are capable of yielding a sound of tension when suddenly put upon the stretch. There may be, therefore, complete suppression of the first sound, notwithstanding that the auriculo-ventricular valves move with freedom, but in such case the valves must be structurally unsound, and the impulse element of the first sound must be masked or abolished.

Da Costa\* suggests that in certain cases in which anæmia is a predominant characteristic, murmur may be due to alteration in the tension of the valves, brought about by excited action of the heart, and communicating to the blood vibration which is intensified by the impoverished state of that fluid, but may exist independently of it.

Oppolzer† holds that a thickened and disorganized valve, even though competent to prevent reflux, may give rise to murmur by the altered character of its vibrations, and that even where regurgitation does take place, and is the principal cause of the murmur heard, the abnormal vibration arising from the structural alteration of the valve acts as a reinforcing cause of murmur.

I cannot subscribe the doctrine of tension-murmurs, and I do not believe that valvular murmurs can be fairly attributed to

\* *American Journal of Medical Sciences*, July, 1869; and *Dublin Quarterly Journal* (abstract) November, 1869.

† *Dublin Quarterly Journal of Medical Science*, November, 1869. Report on Medicine.



this cause, independently of afflux or reflux blood-currents. I have never met with an example of murmur not satisfactorily explicable on other and more rational grounds.

Dr. C. J. B. Williams\* is of opinion that murmur is produced by friction of the blood, moved with a certain degree of velocity and force, against irregular surfaces presented by the surrounding solids. The resistance thus given to the current of blood causes sonorous vibrations in the liquid mass, if the latter be urged onwards with sufficient force and rapidity. The resisting medium may be a thickened or irregular valve, a contracted orifice, or a healthy valve opposing resistance through spasmodic contraction of one of the columnæ carneæ. If the velocity be very low there may be no murmur, even with a rigid or partially ossified valve; hence the occasional subsidence of organic murmurs when the circulation has become languid.

Dr. Williams assumes the operation of two other causes of murmur which are, I think, of questionable efficacy; namely, a *relative* narrowing of the orifices of the heart, consequent upon sudden or abrupt contractions of the organ, as in nervous palpitation from excitement, loss of blood, or from inflammation. In such cases a disproportion of the mass of blood to the orifices arises from the suddenness with which it is forced through. I have never heard a murmur of this kind, with the single exception of that following hæmorrhage; but this is due to a cause totally different from that assigned by Dr. Williams, and as belonging to a different class, namely, that of hæmic murmurs, will be discussed separately under that head.†

The other cause of (to me) a doubtful character, to which Williams attributes murmur, is globular dilatation of the ventricle, by which the orifice of exit, though absolutely healthy, is contracted relatively to the ventricle, and the blood in escaping from it, encounters resistance, and is thrown into a state of vibration. I have not met with an example of murmur due to this

\* *The Pathology and Diagnosis of Diseases of the Chest*, 3rd edition, 1835, p. 193.

† Skoda (*A Treatise on Auscultation and Percussion*, translated from the fourth edition, by Dr. Markham,) is likewise of this opinion, for he says (p. 211) "A contraction of the heart, more forcible and rapid than ordinary, cannot of itself produce a murmur; and, on the contrary, murmurs (non-organic, *scil.*) may exist when the movements of the heart are slow."

cause; I have always found slight roughening or crimping of the aortic valve, or atheromatous disease of the vessel immediately above the orifice, in cases where a systolic aortic murmur of indubitably organic nature had existed; and after death an hypertrophied and dilated ventricle was the most notable organic lesion discovered.\*

Skoda† concedes the correctness of the generally received opinion, that intra-ventricular murmurs are caused "by friction of the blood against the walls of the heart, or against the valves;" but holds that murmur may be also produced within the cavities of the heart by collision between two streams of blood, one of which is moving in a definite direction, whilst the other is moving in the opposite direction, or in the same direction but more slowly; or by the sudden irruption of a stream into a stationary mass of blood.

Doctor Walshe‡ also advocates the sufficiency of unnatural friction between the blood and the surfaces, and likewise of intrinsic collision between two masses of blood in motion, to occasion murmur. The organic alterations which give rise to abnormal friction are, constriction of the orifices, either simple or with thickening; rigidity; calcification of, and wasting or other growths upon, the valves: and those which occasion collision of blood-streams are, simple insufficiency of the valves to close a widened orifice; or incomplete valvular occlusion from organic alteration, of whatever kind, of the valves themselves; from shortening of the chordæ tendineæ; atrophy of the columnæ carneæ; adhesion of the segments of the valve to one another, or to the walls of the ventricle or artery: unnatural communication between the different compartments of the heart, or between any of these and one of the arteries, or between the cavities or the arteries and an adventitious chamber. Polypoid growths in the vicinity of the valves, coagula entangled amongst the columnæ carneæ, mere roughness of the endocardium, and abnormal attachments of one of the chordæ tendineæ, whereby

\* Dr. Blakiston agrees in opinion with Dr. Williams as to the competency of globular dilatation with hypertrophy of either ventricle, without lesion of the arterial orifice, to give rise to murmur.

† *Opus citat.*, p. 201.

‡ *Diseases of the Heart*, 3rd edition, 1862, p. 86-7.

it is thrown directly across the blood-current, are likewise competent to produce murmur under a strong current of blood.

I have not met with a single example which would warrant me in coinciding with Dr. Walshe in the opinion that, in simple hypertrophy of the left ventricle, without narrowing of the aortic orifice, or valvular lesion, "excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur." Neither am I convinced that palpitation of an hypertrophied heart, with or without disease of the papillary muscles, may, by deranging the action of these muscles, cause mitral regurgitation.\*

Dr. Stokes very properly urges† that, for the production of organic valvular murmur a certain force in the propulsive power of the heart is requisite; and that, in the absence of this essential element in the causation of murmur, the latter not infrequently fails to be heard, even where ossific lesion of the valves exists.

Bamberger‡ is of opinion that occasionally murmur arises from insufficient tension and after-vibration of the auriculo-ventricular valves, consequent upon fatty degeneration or simple weakening of the papillary muscles.

It will thus be seen that the doctrine of tension-murmurs, but in various forms, has taken firm hold of the minds of some of the ablest pathologists of the present time.

Nevertheless, I cannot subscribe this doctrine. I do not believe that valvular murmurs can arise from modified tension-property of the valves, irrespectively of afflux or reflux blood currents.

Murmur, like normal cardiac sounds, is produced by vibration; thickening and roughness of the valves operate rather by suppressing vibration; and, if they do not give rise to the eddy of obstructed afflux or efflux, or to that of reflux, they cannot be a cause of murmur.

The primary and essential, though remote cause of murmur,

\* *Opus citat.*, p. 283.

† *Diseases of the Heart and Aorta*, 1854, p. 107.

‡ *Dublin Quarterly Journal of Medical Science*, November, 1869, "Report on Medicine," p. 592.

I hold to be *friction* of the blood-current against the walls or passages where the murmur arises. Friction is a cause of vibration, which is communicated to the walls and to the current, and conducted by the latter in the direction of its flow. Vibration is transmitted by the adjacent solids in accordance with determinate physical laws, being interrupted by dissimilar, and favoured by similar structural constitution of the transmitting medium. Retrograde transmission of vibration, however, through the cardiac or vascular walls, is neutralized by the definite course of the blood-current, whilst progressive or forward transmission through these media is not only favoured, but vibration in them is reinforced, by the blood-stream, itself in a state of vibration from forcible impact at the seat of friction. There is one exception, rather apparent than real, to the non-transmission against the current, of vibratory pulsation of the solids; namely, that which is afforded by regurgitant apex-murmurs arising from reflux at the auriculo-ventricular openings. These murmurs are loudest at the apex, although the current by which they are produced sets in the opposite direction, or towards the base of the heart. But the chordæ tendineæ, owing to their intimate connexion with the mitral and tricuspid valves, their eminently-vibratile properties, and isolated position in the cavities of the ventricles, must receive and communicate to the muscoli papillares, and through these to the ventricular walls at the apex, vibrations originating in the valves. The peculiar arrangement of these tendons, moreover, that, namely, by which the greater number of them supplies filaments to two adjacent and opposed valve-segments, renders their *direct* interference with a current of reflux at the auriculo-ventricular orifices unavoidable; they thus, in a measure, create and directly receive the vibrations of that current, and, of necessity, transmit them through the papillary muscles to the apex.

That friction as between a fluid, whether liquid or aeriform, and a solid surface of contact, irrespectively of the regularity of that surface, is capable of yielding vibration audible as murmur and tangible as tremor, is sufficiently illustrated by the passage of water under strong pressure through hose or metallic pipes upon the one hand, and by the phenomena of whistling and



playing upon wind-instruments on the other. In both these cases alike, fluid-friction against the sides of the tube, the lips of the performer, or the mouth-piece of his instrument, becomes a cause of intrinsic molecular commotion, or vibration, in both the fluid and the solid medium; perceptible in the latter to the sense of touch as *tactile vibration*, *tremor*, or *fremitus*; and in both, sensible to the ear as sound more or less harsh, or *murmur*.

Both these phenomena are likewise exemplified in sonorous rhonchus, which, when loud, is always associated with fremitus.

Murmur and fremitus, though due to a common cause, are not always associated, because fremitus requires for its production stronger and coarser vibration than does murmur.

Hence fremitus is a much less constant and invariable phenomenon than murmur; and hence likewise it is, that whilst both are frequently associated, whenever that association is dissolved by disappearance of either phenomenon, fremitus is that which ceases to be manifested.

Within the last ten years much has been done experimentally, and written, especially in France, with the object of elucidating the phenomena of cardiac and respiratory murmurs. The results obtained have tended, in a singular manner, to confirm those of Sir Dominic Corrigan already referred to; and to establish the general correctness of the views as to the cause of murmur, and the laws of its propagation, expressed by that eminent physician so long ago as 1829.

The writers to whom I especially refer are M.M. Chauveau,\* Boudet,† and L. Bergeon.‡ The vascular bruit has been shown, in the opinion of these writers, to be due to an intrinsic commotion in the circulating fluid, designated "*veine fluide*," by Savart. M. Bergeon says: "Whenever a liquid flows from a reservoir,

\* "Études Pratiques sur les Murmurs Vasculaires, ou Bruits de Souffle, et sur leur valeur Séméiologique," *Gazette Médicale de Paris*, 1858, p. 247. For this and the subsequent references, as well as for an able summary of this subject generally, I am indebted to the writer of Article ii., "On the Physical Theory of Murmurs, vascular, cardiac, and respiratory," in the *British and Foreign Medico-Chirurgical Review* for July 1873.

† "Recherches Physiologique sur le Mécanisme des Bruits Respiratoires," *Gazette Hebdomadaire*, 1863, p. 789.

‡ *Des Causes et du Mécanisme du Bruit de Soufflet*, Paris, 1868; and *Théorie des Bruits Physiologiques de la Respiration*, Paris, 1869.

through a vertical or horizontal orifice, the stream takes the form of a jet, to which Savart gave the name of 'fluid vein.'"

"The 'fluid vein' is essentially composed of two parts, the first calm, transparent, like a stem of crystal; the second agitated, without transparency, but so far possessed of regular form, that it can be seen to be divided into a certain number of elongated swellings (of which the maximum diameter is always larger than that of the orifice), separated by narrower portions. This nodal appearance of itself shows that the liquid vibrates, and by means of the electric light Savart showed that the flow is not really continuous. The 'fluid vein' in part consists of separate drops of the fluid, succeeding one another at regular intervals, each drop changing its form in passing from point to point."

The production of a "fluid vein," or vibrating axial current, whether in a liquid or a gas, depends essentially upon its transmission with a certain force through a narrow orifice into a larger space beyond, whether that space contain air or liquid. And in this relatively larger or (in regard to the direction of the current) distal space alone the "fluid vein" or sonorous vibration is generated. This, as shown by Bergeon, and as long before correctly stated by Corrigan, is due to the different pressure experienced by the fluid on opposite sides of the constricted orifice. To the same conclusion leads an experiment performed by Chauveau on a pithed horse, artificial respiration having been kept up; from this and other experiments he deduces the following general law:

"Bruit de souffle is always produced by the vibrations of an intra-vascular *veine fluide*, and such a *veine* is constantly formed whenever the blood passes with a certain force from a narrowed into an actually or relatively dilated part of the circulatory system." But manifestly, under this law, something besides intra-vascular narrowing may produce a "fluid vein," and something other than "fluid vein" may produce *bruit de souffle*.

Roughness of the intra-cardial and intra-vascular surface as a cause of murmur, has been summarily set aside by Chauveau, but as I think, on insufficient evidence,

On the same principle, that of a "fluid vein," Chauveau like-

wise explains anæmic murmurs at the base of the heart, and in the great veins of the neck.

In anæmia there is a general reduction in the volume of the blood; the chambers and orifices of the heart, and the blood vessels generally, with two exceptions, accommodate their diameter to the reduced volume of blood. The exceptions are: (*a*) the aorta and pulmonary artery, which, owing to the absence of the contractile and the preponderance of the elastic element in their walls, cannot reduce their diameter proportionately to that of the current passing through them. Hence a "fluid vein" and murmur of exit. (*b*) The roots of the innominate veins are fixed and kept permanently dilated by the cervical fascia, which not only ensheaths them, but is connected with the sternum, clavicle, and first rib. Hence, whilst the jugular and subclavian veins above accommodate themselves to the reduced diameter of their respective currents, the commencing portion of the innominate veins, being incapable of a reduction of calibre, give origin to a "fluid vein," and a venous hum, or *bruit de diable*.

This is a most ingenious and apparently a satisfactory explanation of venous murmur. Against Chauveau's theory, as applied to the heart, the apparently insuperable objection may be urged, that it necessarily implies a murmur of diastole at the auriculo-ventricular orifices in the normal state of the heart. Corrigan anticipated this objection, by alleging that normally such a murmur is actually but faintly audible at the points mentioned. But, in point of fact, no such murmur can be detected in the physiological state.\*

Murmurs are governed by the laws of ordinary sound in regard to their pitch, quality, and intensity.

The *pitch* or key of a murmur is determined by the number of sonorous undulations occurring within a given time; the greater the number of such, the higher the pitch; and conversely. Some murmurs are of so low a pitch that they scarcely form a continuous sound, whilst others, on the contrary, have a pitch so high that they are musical in character. As a rule, to which I

\* Further, the remarkable difference in the *quality* of murmurs would not be explicable on this theory.

am not acquainted with a single valid exception, very high pitched, and hoarse or low pitched murmurs, are of organic or of thromboid origin, and therefore of less favourable significance than those of an intermediate key.

The *quality* of a murmur, expressed by its harshness or the opposite, depends upon the abruptness of the vibrations to which it is due. Harsh grating murmurs are the result of the attrition of two uneven and dense surfaces against one another, or of a strong current upon solid, rugged, and projecting points of surface. Soft murmurs are caused by the movement of currents over smooth and even surfaces; and therefore, whilst they may indicate formidable valvular disorganization, they cannot be regarded as evidence of atheromatous or calcareous transformation.

I cannot agree with Hope in holding that harshness depends upon the configuration of the orifice, rather than upon the character of the disorganizing medium. Nor can I admit with him, that "osseous disease" of the cardiac valves or vascular walls, if covered by the lining membrane, may give rise to bellows murmur; and that, on the contrary, fibrous and fibro-cartilaginous structures may give origin to rasping and sawing murmurs.

The intensity of a murmur is directly as the strength of the primary impulse, the proximity of the seat of origin to the ear, and the conducting properties of the intermediate structures. Proximity influences the loudness or distinctness of a murmur; but it certainly is an error to assert, as Hope has done, that it modifies, much less determines, the pitch of a murmur. It has been shown experimentally by Biot, that distance has no influence on pitch. If this were not so, musical notes of different pitches, heard at a distance, would run into one another and be out of harmony; but such is notoriously not the case. Dr. Walshe, writing on this subject says,\* "The mere distance of the site of production of murmurs from the surface, can, in theory, have no direct influence on their pitch. The same physical and dynamical conditions will generate sounds of the same pitch, whether they be close to, or as far as possible from, the chest-wall."

Murmurs have been classified according to their position, their

\* *Opus citat.*, p. 90.



rhythm, relationship to the normal sounds, pitch, and quality; and a complete definition or description of a murmur must include a reference to each of these characteristics. This is a matter of some moment in regard to intelligible description; and it were much to be desired that this, or some other equally convenient formula for the designation of cardiac murmurs, should be universally adopted by physicians. Till a universally recognized standard of this kind be established, laxity of description, and consequent confusion and misinterpretation will continue to be, as at present, unavoidable.

The order in which I have just mentioned the several characteristic features of a murmur is the analytical. The synthetical method of description, however, will be found more convenient as applied to any particular murmur, as is practically found to be the case in regard to other matters, in specifying one of a multitude of objects of the same kind. I shall revert to this subject after having treated of the different characteristics of murmurs as above sketched.

The site or *point of maximum intensity* of a murmur is of great importance in regard to differential diagnosis. Murmurs have been divided according to their site into *apex murmurs*, and *basic murmurs*; and each of these groups has been subdivided into left and right. Thus there are, in the order of their frequency, *left and right apex murmurs*, and *right and left basic murmurs*.

The determination of this point of maximum intensity of a murmur is a purely acoustic operation, in the performance of which, however, great readiness and precision are acquired by practice and close attention.

The *rhythm* of a murmur has been interpreted by the majority of writers on diseases of the heart, as having reference only to those periods of the cardiac cycle which are covered by the normal sounds, and is by medical men generally so regarded. Nevertheless, it is an unwarranted limitation of the meaning of the term, because it implies that during the intervals between the sounds, or the short and the long pause respectively, murmur is never heard.

That murmur is, however, occasionally and not unfrequently

audible during the normal periods of silence, there can be no longer any doubt; and furthermore, that murmurs of this particular rhythm have, in regard to each of the two pauses, a specific significance of great diagnostic value, I am quite convinced, and undertake in the sequel to shew.

Gendrin\* seems to have been the first to recognize this important distinction, to which, indeed, his subdivision of the normal periods of silence (page 82) led him by logical necessity.

Gendrin's classification of murmurs on this basis is, however, not only eminently unpractical, but highly erroneous, as will appear further on; but to him, nevertheless, belongs the merit of having been the first to recognize the existence of intersonal murmurs, and to introduce corresponding designations, even though somewhat overdrawn, and in many instances incorrectly applied.

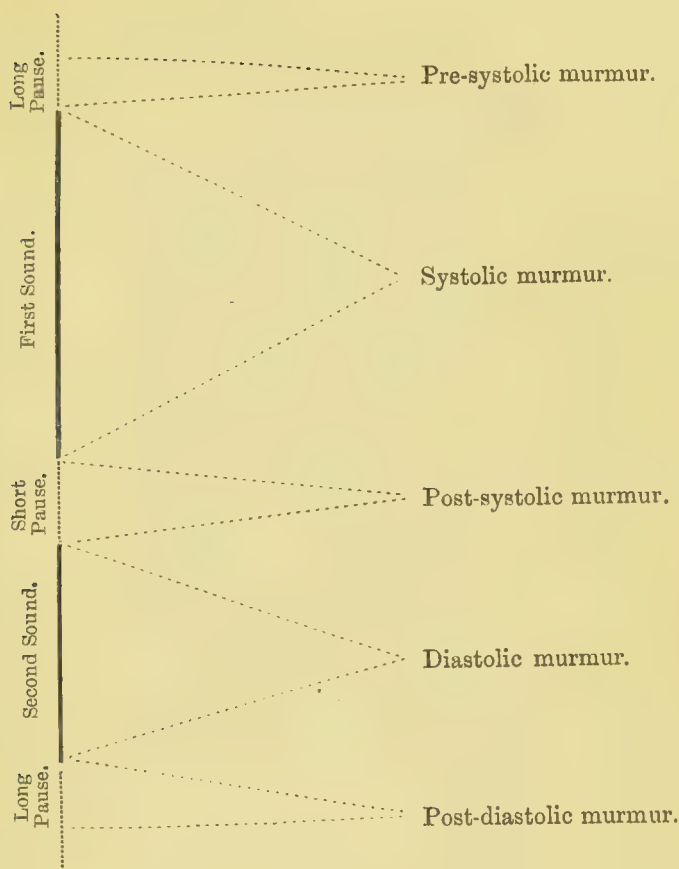
Doctor Walshe not only recognizes the existence of murmurs falling within the periods of cardiac silence, as distinguished from those of sound, but proposes a much more euphonious nomenclature. He says, "But they [murmurs] are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, or post-systolic; pre-diastolic, diastolic, or post-diastolic."†

Many years ago, before I had the pleasure and advantage of reading Dr. Walshe's valuable work, I had, from clinical study, perceived the necessity of admitting intersonal murmurs, and actually used the designations I am happy to find Dr. Walshe has anticipated me in proposing. The appreciation, by two independent observers, even though at different periods, of a special and distinctive morbid phenomenon, and referred by them to the same cause, is, *pro tanto*, evidence of the accuracy of their respective observations.

At the meeting of the British Medical Association at Oxford in 1868, I read a paper in which I set forth my views on this subject, and gave cases in illustration. The subjoined diagram will be found to render this classification simple and intelligible:

\* *Leçons sur les Maladies du Cœur*, 1841-42.

† *A Practical Treatise on the Diseases of the Heart and Great Vessels*, 3rd edition, 1862, p. 85.



The preceding classification of cardiac murmurs may seem to involve unnecessary refinement; that is, to imply distinctions not actually to be met with in disease. I am aware that many highly respectable pathologists, and a few of special authority on this subject, hold that if it be not quite impossible in practice to draw the distinction between *sonal* and *intersonal* murmurs as above indicated, at least no information of value, in regard to diagnosis or treatment, can be deduced from it. To this objection, to which I pay all due respect because of its authorship, I shall give a very simple answer. If the distinction be founded in nature, and murmurs of the special rhythm of those named *intersonal* be of actual, even though rare occurrence in disease, then it is sufficiently warranted, and needs no further justification, quite irrespectively of its imputed value as an aid to differential diagnosis.

Of the existence and special diagnostic value of at least one, and that the typical form of this class of murmurs, namely, the presystolic, doubt is no longer entertained by anybody not altogether impenetrable to the light of progress. Yet less than thirty years ago the existence of a murmur of this special rhythm was utterly unknown, and notwithstanding the suggestive intimation of its distinct identity given by Walshe in England, and the more positive affirmation of Markham and Gairdner to the same effect, presystolic murmur had not a place assigned to it in the established semeiology of English medical literature till 1867, when Doctor Peacock fully admitted its separate entity and special significance,\* and attributed his conversion to Dr. Gairdner's writings and mine on this subject.

Bouillaud in 1835† regarded the murmur of auriculo-ventricular constriction as diastolic in rhythm, as may be inferred from a passage in his *prolégomène*, in which, whilst discussing multiplication of the sounds of the heart, he adduces an example of quadruple sounds; the first associated with slight murmur, the second and third sharp and harsh, and the fourth represented by a bellows murmur. The second and third he regarded as a reduplicated second sound, "as if the ventricle had not the power to fill itself, save in two movements, or in two efforts." Herein Bouillaud implies, what he elsewhere, as already shown, distinctly states, that in his opinion, the second sound is caused by the passage of blood into the ventricles; and further, that the murmur of auriculo-ventricular constriction, if such exist, must be diastolic in rhythm, or one accompanying or replacing the second sound of the heart.

Gendrin in 1841‡ subdivided the periods of cardiac silence, as previously stated at length, into the *péridiastole*, and *présystole*, the *périsystole* and *prédiastole* respectively; but that he misapprehended the precise rhythm of at least one, and that the most important of the murmurs which should correspond to these sub-divisions, will be manifest from the following references to his work. He says: "When the column of blood, in

\* *British and Foreign Medico-Chirurgical Review*, No. lxxx., October, 1867.

† *Opus citat.*

‡ *Leçons sur les Maladies du Cœur.*



passing from the auricles into the ventricles, encounters rugosities at the auriculo-ventricular orifices, we hear a *prédiastolic* murmur. If the rugosities extend to the free edge of the valves, this murmur is prolonged to the second sound, and ends with it. If the alteration occupy exclusively the free edge of the valves, without engaging their attached border, or the auriculo-ventricular orifice properly so called, the murmur *commences only with the diastole, and terminates with it*; it is then *diastolic*. When the murmur is *prédiastolic* we are warranted in suspecting that narrowing of one of the auriculo-ventricular orifices exists. We may positively affirm that such is the case, if there exist at the same time doubling of the second sound (*percussion diastolique*). The blood occupies a longer time in traversing the narrowed auriculo-ventricular orifice, and the murmur is the more prolonged, the greater the contraction of the orifice is."

"*Prédiastolic* murmurs, which are produced by obstacles to the free and ready passage of the blood through the auriculo-ventricular openings, are heard with greatest distinctness at the middle of the vertical diameter of the precordial region; they are lost at the base, and towards the apex of the heart."

"Systolic, péricystolic, and *prédiastolic* murmurs of endocardial origin are often combined. Thus, if rugosities exist on both surfaces of the mitral valve, and are very prominent, a *prédiastolic* murmur may be heard coincidently with the passage of blood through the auriculo-ventricular orifice, and another, a systolic murmur, produced when the wave of blood strikes against the surface of the valve during ventricular contraction; these two murmurs are likewise met with when the mitral valve is inadequate."

He also writes: \* "Auriculo-ventricular narrowings most usually have their seat at the summit of the valves, then coherent; thence results a cone-shaped infundibulum, at the bottom of which is found the contracted orifice, forming a sort of muzzle with two or three lips. If, in the bottom of this infundibulum with thick and indurated walls, inequalities, or small warty and fig-like tumors be found, *the diastole is preceded and accompanied* by a vibratory thrill which is perceived in greatest in-

\* *Opus citat.*, p. 130.

tensity at the middle of the vertical diameter of the heart, on its left border, if the lesion be, as most commonly it is, at the left *auriculo-ventricular orifice*.

"The *prédiastolic* vibratile thrill, which is produced in such cases, is always feeble, at least as regards the mechanical transmission of the vibrations to the walls of the chest.

"We often meet with the valvular lesion just indicated, as a cause of *prédiastolic* vibratile thrill, to coincide with different lesions, likewise previously described, and to the presence of which is to be attributed systolic vibratory thrill. In this case there are two thrills separated by a period of silence, *which measures the length of the interval between the diastole and the systole*."\*

The extracts above given from Gendrin's book show conclusively that he has no claim to the merit of having recognized the diagnostic significance of presystolic murmur, because he has quite mistaken for it a murmur of different origin and rhythm, namely, one associated with the second sound of the heart, and therefore separated in time from the presystolic murmur by the greater portion of the long pause. Nor has Hope any better claim to the distinction of having recognized, much less discovered, the presystolic murmur, as will appear from the following extracts from his great work.

He says:† "Will it be said that the auricular contraction, previous to the ventricular, should create a murmur? I have looked for it carefully, and have only once been able to suspect it, without being able to assure myself of its existence. Theoretical reasoning seems to countenance this result of observation; for, as the auricular systole is slight, the quantity of blood injected by it is not considerable, and as the ventricle is already *full*, it cannot admit that extra quantity necessary to bring it to the state of distension, without offering a resistance to its ingress, which must greatly retard the force and velocity of the current; a force, indeed, which can never be great, because the auricles are not only weak muscles, but are unsupported by valves behind."

\* *Opus citat.*, p. 111. The italics, I need scarcely observe, are not in the original.

† *A Treatise on the Diseases of the Heart and Great Vessels*, 3rd edition, 1839, p. 79.

Discussing (p. 80) diastolic murmur at the tricuspid orifice, he says: "It is so rare that (abstracting *pulmonic and aortic regurgitant murmurs, with which it is apt to be confounded*) I am not satisfied that I have ever met with an instance of it." "The reason assigned for the rarity of *diastolic* murmurs in the contracted mitral valve, namely, the feebleness of the current of blood, applies equally to the tricuspid."

He describes (p. 78) murmur of mitral contraction as rare, "*diastolic*," and liable to be confounded with that of aortic reflux, from which, he says, it is to be distinguished only by its being confined to the apex.

Finally, in reference to the differential diagnosis of aortic regurgitation, he writes: "The murmur accompanying the *second sound* I am inclined to attribute, perhaps entirely, to the same (viz., inflammatory) constriction affecting the *auriculo-ventricular valves*, or, I should have added, occasioning *patency of the sigmoid valves*;" and again (p. 74-5) "It may be diagnosed from mitral *diastolic*, always feeble, but inaudible at the aortic valve, *as they both occur during the diastole*, by its being loudest at the aortic orifice, though prolonged towards the apex."\*

Thus Hope states, *passim*, that the murmur of mitral obstruction is diastolic in time, and that by its rhythm it is liable to be confounded with aortic and pulmonic reflux murmurs. Hence it is clear he failed to identify the presystolic murmur of mitral constriction, which, in its ordinary form, occupying not the initial but the terminal portion of the long or diastolic pause, is in no manner associated with the diastolic or second sound of the heart; and therefore, not in the remotest degree liable to be confounded with the regurgitant murmur of aortic or pulmonic inadequacy.

A *veritable* apex diastolic murmur is undoubtedly "rare," as Hope describes it. I have met with only two examples of such murmur, and Doctor Stokes informs me he has met with another. In one of my cases I had the advantage of an autopsy, the particulars of which I shall give in connexion with a more lengthened discussion of this subject in a future page.

\* The italics in the preceding quotations, with the exception of the word "*full*," are not in the original.

Skoda, discussing the physical diagnosis of mitral constriction, states as follows: \* “*In the left ventricle, during its diastole, a sound unaccompanied by a murmur (second sound) indicates that there is no constriction of the left auriculo-ventricular opening, and that the blood, in passing from the left auricle into the left ventricle, does not flow over any roughened surface.*

“*A murmur accompanied by a sound, or a murmur alone, indicates either constriction of the mitral orifice, with roughness of the narrowed surface of the canal, or the presence of rough eminences upon the auricular surface of the mitral valves, unaccompanied by constriction of the opening.*”

“If the left auriculo-ventricular opening be constricted (and this condition is almost always associated with defect of the mitral valves), the *diastolic murmur* is generally loud and prolonged, the systolic being weak and of short duration,” etc.

Thus he lays it down that a second sound in the left ventricle, unconnected with murmur, is conclusive evidence against mitral constriction or roughness of surface; and conversely, that a murmur accompanying the second sound, or superseding it, is positive evidence of mitral narrowing; and finally, if the mitral orifice be narrowed, as distinguished from simple roughness of surface, the murmur with, or replacing the second sound, is loud and prolonged.

Doctor Stokes says: † “In the ordinary cases of mitral murmur we cannot say whether the murmur is “constrictive” or “regurgitant,” or constrictive and regurgitant; and we must reject a large proportion of descriptions of phenomena which, although the changes they are supposed to indicate be familiar to anatomists, are themselves of doubtful value.”

Dr. Bellingham states: ‡ “Disease of the mitral valve or orifice, causing an impediment to the current from the left auricle into the left ventricle, is sufficiently common; but a *diastolic* murmur from this cause is excessively rare; so rare that the most excellent observers have never heard it. Indeed, the current from

\* *A Treatise on Auscultation and Percussion*; Markham's translation, 1853, pp. 232-3-4.

† *Diseases of the Heart and Aorta*, 1854, p. 181.

‡ *On Diseases of the Heart*, 1857, part ii., p. 383-4.



the auricle into the ventricle is generally too feeble to develope one, and this condition of the mitral valve and orifice is characterized rather by a diminution in the intensity of the *second sound* than by a murmur.

"I cannot help thinking that, in the cases in which a murmur from this cause is said to have been heard, the murmur of mitral regurgitation was mistaken for it, which sometimes, instead of accompanying the ventricular systole and the first sound, *follows* it, and thus obscures the second sound of the heart."

No doubt, a diastolic murmur from this cause is excessively rare. I also freely admit that the current of blood from the auricle into the ventricle is too feeble under ordinary circumstances to develope a murmur, save towards the termination of auricular systole, or during its *momentum*, when a murmur not only may occur in mitral contraction, but as a matter of fact is very rarely absent in cases of that lesion.

Occasionally an example is met with of murmur immediately following the first sound, or postsystolic murmur; but such a murmur belongs to, and is distinctive of, mitral regurgitation, not mitral contraction, which may or may not be likewise present; it extends into the short or systolic pause, and may even be so far prolonged, though it rarely is so, as to touch the second sound, in which case alone it could be regarded as in any sense diastolic. But whether it be or be not so prolonged as to give rise to a mistake in regard to its proper rhythm, under no conceivable circumstances can it be confounded with the murmur of mitral obstruction or constriction, which is *never* exclusively diastolic, in the sense of being synchronous with the second sound; *never* engages any portion of the short or systolic pause; *always* precedes the first sound; and *always* engages the long or diastolic pause.

Dr. Ormerod, discussing some novel forms of murmur,\* writes: "A murmur, audible at or near the apex only, accompanying *the first sound*, rising in intensity with the continuance of the first sound, and ending suddenly with a snap at its closure, admits of more than one interpretation. Its seat is unquestioned: the mitral or the tricuspid orifice. But is it due to *obstruction* to

\* *British Medical Journal*, August 6th, 1864.

the flow of blood from the auricle to the ventricle during the auricular systole, or to imperfection of the auriculo-ventricular valve, allowing regurgitation during the contraction of the ventricle? To the first explanation we may object, that a direct mitral murmur, such as is generally admitted to be of this nature, is quite unlike the murmur under consideration; it is soft, and closely resembles the murmurs of sigmoid regurgitation. Such a murmur is very rare; while the murmur under consideration is not at all uncommon. It may be questioned, too, whether the contraction of the auricles, slight and momentary as experimentalists report it to be, is capable of producing so loud a sound. To the second explanation I do not see that there is any insuperable objection."

It is manifest from this extract that Dr. Ormerod, so late as 1864, regarded the rhythm of direct or mitral obstructive murmur as systolic, or "accompanying the first sound;" and from the objections which he urges against this hypothetical view, and which have reference to the quality and the rarity, not to the rhythm of the murmur, it is equally clear that he was unacquainted with the distinctive characteristics of presystolic murmur. The quality (softness) which he assigns to it is, moreover, not that which really belongs to it.

Dr. Blakiston says:\* "The chief physical sign of mitral obstruction is a *diastolic* murmur; it is rarely, however, that such a murmur is engendered, because the size of the auriculo-ventricular opening is so large," etc. Here the rhythm of the murmur is likewise mistaken as being "diastolic," an error which, by fixing attention exclusively upon the second sound, may lead either to non-observance of the murmur where it actually exists, or to non-recognition of its true nature even when detected.

Doctor Walshe states:† "A diastolic murmur of maximum force, *immediately above and about the left apex*, and conducted in the same direction, though less extensively, as systolic murmur of the same seat, indicates *obstructive* narrowing of the mitral orifice, or simple roughness of the auricular surface of the mitral valve; or both states combined.

\* *Diseases of the Heart*, 1865, p. 246.

† *Diseases of the Heart*, third edition, 1862, p. 103-4.

"This murmur is commonly spoken of as diastolic in rhythm ; but, in point of fact, it is rather postdiastolic, or *præsystolic*, than precisely coincident with the diastole."

Further on he adds\* : "I have already stated that the rhythm of this murmur is rather postdiastolic, or *præsystolic*, than actually diastolic."

It appears from these extracts that Dr. Walshe had a not very definite, but still, *pro tanto*, a correct appreciation of the rhythm of this murmur, and that he was, beyond all doubt, the first in this country to point it out.

Sir William Gull fully admitted† the diagnostic value of pre-systolic murmur. Yet three years later I find Dr. Tanner stating as follows:‡

"A diastolic murmur most distinct from the centre of the sternum (on a level with the third intercostal space) upwards towards the base, with a jerking pulse, is indicative of aortic regurgitation ; while a *diastolic murmur*, most distinct from the fourth left intercostal space, onwards towards the apex, with an irregular small pulse, is the result of *mitral obstruction*." And Dr. Niemeyer in his celebrated work§ lays down rules for the identification of this murmur as follows, viz. : "Upon auscultation we almost always hear a long-drawn murmur at the apex during diastole." He adds, "In addition, we can, of course, hear the *second sound* propagated from the arteries, unless the murmur be too loud."

Manifestly Dr. Tanner failed even to identify the murmur to which so much significance attaches ; for he describes it as diastolic in time, and also as being associated with an irregular small pulse, a symptom which, though met with in connexion with mitral stenosis in its last stage, or when death is imminent, most certainly does not belong to that lesion at a time when differential diagnosis is of any consequence to the patient.

Niemeyer, whilst associating with this murmur "presystolic purring," strangely and inconsistently describes it as "diastolic," and associates it with the "second sound."

\* *Opus citat.*, p. 372.

† *Medical Times and Gazette*, January 27th, 1866.

‡ *The Practice of Medicine*, sixth edition, 1869, vol. i., p. 619.

§ *A Text Book of Practical Medicine*, second edition, 1869, vol. i., p. 356.

Dr. Aitken says:\* "The murmur indicative of obstructive narrowing of the mitral valve is a *ventricular diastolic murmur* heard in maximum force *immediately above and about the left apex.*"

The late eminent Professor Trousseau likewise described this murmur as *diastolic* in time, as shown by a passage in his recently published lecture on "Organic Diseases of the Heart."†

To M. Fauvel belongs the merit of having been the first to identify the presystolic murmur, and fully appreciate its pathological significance, as will appear from the following quotations from his memoir published in 1843.‡ He gives Beau the credit of having been the first to expose the fallacy of attributing a diastolic rhythm to the murmur of auriculo-ventricular constriction, by declaring that "There is not on record a single well-authenticated example of diastolic murmur at the apex in connexion with narrowing of the auriculo-ventricular orifice."

In 1843, Fauvel observed in the person of a discharged soldier an intense bruit de râpe preceding the first sound, ending with it, and loudest at the apex and to the left. Four new cases followed, and of these three were fatal.

Case 1, a female, aged fifty, the subject of a chronic cerebral affection; strong impulse and extended precordial dullness. A strong bruit de râpe loudest at the level of the fifth rib, to the left of the nipple, and growing faint towards the right; commencing in the long pause, and ending at the instant of the first sound; pulsation intermittent; pulse small and irregular, and no cedema. Death from the cerebral affection. The left auriculo-ventricular orifice was contracted to the size of the tip of the middle finger, and cartilaginous and warty on the auricular aspect; slight hypertrophy of the left ventricle, but no dilatation. In reference to this case he remarks: "It was of importance in this, that it exhibited a considerable narrowing of the auriculo-ventricular orifice, coinciding with an abnormal bruit associated with the first sound in such a way that it commenced almost in

\* *The Science and Practice of Medicine*, second edition, vol. ii., p. 717.

† *Lectures on Clinical Medicine*, New Sydenham Society, vol. iii., 1870, p. 413.

‡ "Mémoire sur les signes stéthoscopiques du Rétrécissement de l'Orifice Auriculo-ventriculaire gauche du Cœur," *Archives Générales de Médecine*, tom. i., 1843.



the middle of the long pause, and ended at the instant when the first sound was heard."

Case 2 exhibited a rough murmur commencing before the normal first sound and ending with it, so as to impart to it a very hoarse pitch (*fortiment enroué*). On examination after death the mitral was found contracted to the size of the little finger.

Case 3 yielded a bruit de râpe preceding and covering the normal first sound at the apex and to the left. There were likewise an apex and an aortic postsystolic murmur. The mitral orifice was found contracted to the size of the little finger, and the valves inadequate; the aortic orifice was contracted, and rough by excrescences. He concludes, correctly, that the presystolic murmur at the apex was due to mitral obstruction, and the postsystolic murmur to mitral reflux. The three last-mentioned patients were females.

Case 4, a man aged thirty-two. "A bruit de râpe, which commenced in the long pause an instant after the second sound, and ended with the first." Left hospital improved.

Case 5, a man of fifty. "An abnormal bruit at the apex and to left, commencing a little before the first sound, and imparting to it a hoarse character. There was likewise the murmur of aortic reflux. On *post mortem* examination the mitral orifice was found barely to admit the index finger, and the aortic valves were rough and inadequate." He adds: "From all this it follows, as the first consequence, that a bruit de râpe localized at the apex and to the left, and immediately preceding the normal first sound, may be the only morbid sound corresponding to a very considerable narrowing of the left auriculo-ventricular orifice, without inadequacy."

The accuracy with which the premises are above stated, and the justness of the conclusion drawn from them, leave nothing to be added. He designates the murmur as "presystolic," a term for which he is indebted to Gendrin. But as already stated, Gendrin used it with a different meaning. He says "It is the most probable sign of narrowing of the mitral orifice, but I don't pronounce it a certain sign, because the small number of facts on which the conclusion is based does not admit of its being

otherwise regarded than provisional, and as requiring the sanction of fresh observations." In reference to this very modest declaration it may be said that the confirmation intimated as necessary has been since amply supplied,

M. Hérard, ten years later, questioned two of Fauvel's observations, because, forsooth, the murmur is described "as commencing soon after the second sound." He casts doubt upon the entire doctrine of his distinguished compatriot, and strenuously labours, no doubt in good faith, to bring back the state of chaos which he had dissipated. Thus, he maintains that mitral narrowing may give rise to a *systolic*, a *pre-systolic*, or a *diastolic* murmur, and that in old subjects it may be unrepresented by murmur of any kind.\*

Hérard gives a list of cases in which an opportunity for correcting the diagnosis by dissection was afforded, and another in which the diagnosis remained uncertain. The former I shall give in summary.

Case 1, a female aged twenty-four; a rough systolic murmur synchronous with the carotid pulse. On *post mortem* examination the mitral orifice was found reduced to the size of the little finger, with smooth and thick edges, but the valve was competent as proved by the water test. All other openings and valves healthy. The circumstances above mentioned warrant the inquiry whether the rhythm of the murmur was not misapprehended? It must have been presystolic, however masked.

Case 2, a man aged thirty-seven. Double apex murmur (systolic and diastolic) and fremitus. The mitral orifice was found reduced to the size of the little finger, funnel-shaped and rigid.

"Diastolic" murmur in this case was, I suspect, really post-diastolic, and may be explained by the extreme narrowing which existed; owing to which the *passive* disgorgement of the left auricle at the commencement of ventricular diastole, and therefore synchronous with the second sound, may have given rise to murmur extending beyond it. When the auricle had

\* *Archives Générales de Médecine*, tom. ii., 1853. For this and many of the succeeding references I am indebted to Dr. Hilton Fagge, whose valuable memoir in the *Guy's Hospital Reports*, third series, vol. xvi., is indeed a repertory on this subject.

partially unloaded itself murmur ceased, to return with the systole of the auricle at the end of the long pause.

Case 3, a man aged thirty-five. Systolic murmur at base, and double murmur at apex. The aortic valves were found thickened, and the aorta rough by atheromatous plates. The mitral orifice was contracted, and one segment of the mitral valve was rigid and held open by an ossified papillary muscle and tendinous chord.

Case 4, a man. Diastolic murmur at the apex. Fibro-cartilaginous transformation and narrowing of the mitral opening were found.

Case 5, a woman aged twenty-two. Double apex murmur (systolic and diastolic) and *fremitus felinus*. The mitral opening was found narrowed to the size of the little finger, rigid, and funnel-shaped. The tricuspid orifice was in the early stage of similar change. The observations made in reference to Case 2 may be likewise applied to this.

In the discussion of Hérard's memoir M. Beau reiterated his peculiar doctrine as to the rhythm of the different movements of the heart, maintaining that ventricular diastole occurs in "le premier temps," and no portion of it in "le second temps," save in the case of inadequacy of the aortic valve, when it is of necessity prolonged into the period of the second sound. He affirmed, moreover, that diastolic murmur at the apex is always due to aortic reflux, and *never* to mitral narrowing.\*

The entire of this doctrine is now very properly regarded as heretical, the justly high authority of M. Beau notwithstanding.

In 1859 M. Racle† doubted the possibility of identifying the peculiar rhythm of presystolic murmur in practice, and inclined to regard it as systolic, and in 1862 Durozier sneered at it as a myth.‡

Three years anterior to the last-mentioned date, however, Dr. Austin Flint of Philadelphia had in effect admitted the identity and distinctive character of presystolic murmur;§ he designates

\* *Archives Générales de Médecine*, 1854.

† *Traité de Diagnostic Médical*, 2ème ed. 1859, p. 290.

‡ *Archives Gén.* ser. v., tom. xx.

§ *Pract. Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart*, Philadelphia, 1859.

it as diastolic, but adds that it is more strictly presystolic. A few years later he affirms, in more positive terms, its distinctive rhythm, and more accurately describes its quality.\*

"Mitral direct murmur" he says "precedes the first sound. The mitral direct current of blood, therefore, occurs just before the ventricular systole; it continues up to the ventricular systole, and must of course cease when the ventricles contract. The contraction of the ventricles causing the first sound of the heart, it follows that the mitral direct current caused by the auricular contraction must take place just before the first sound, that it must continue to the first sound, and that it cannot continue an instant after the first sound.

"In my work on *Diseases of the Heart* I have said that this murmur (presystolic) is generally soft. My experience since that work was written has shown me that this statement is incorrect.

"I should say that whilst the mitral systolic murmur is much more frequent in its occurrence than the mitral direct, the former, indeed, being the most common of all the murmurs, the mitral direct is observed quite as often without as with the mitral systolic. Between the mitral direct and the mitral systolic (when they concur) occur the apex beat, first sound, and carotid pulse. (??)

"The murmur, however, may be produced by the flowing of the current of blood over a roughened surface, without contraction of the aperture. This is undoubtedly rare.

"Is this murmur ever produced without any mitral lesion? One would, *a priori*, suppose the answer to this question to be in the negative. Clinical observation, however, shows that the question is to be answered in the affirmative." (??) He then goes on to state that he had met with two cases characterized by well-marked mitral direct murmur; in one of which no mitral lesion whatever was found after death, and in the other the lesion was insignificant. In both the aortic valves were obstructive and inadequate. He proceeds to state that when, owing to aortic reflux, the left ventricle gets filled prematurely, the auriculo-ventricular valves are approximated and

\* *American Journal of the Med. Sciences*, vol. xlv., July, 1862.



oppose influx from the auricle, causing thereby direct mitral murmur. To this theory I object: such approximation occurs in the normal state of the chambers and valves, and yet no murmur occurs; no such murmur occurs in simple aortic reflux; and finally, the pressure of accumulation in the ventricle would not present sufficient resistance to the entering current to cause direct murmur.

Dr. Flint is of opinion that a pure diastolic mitral murmur may occur during the passive entrance of blood from the auricle into the ventricle, and thinks he has met such cases.

This remark is eminently just and confirmed by recent observations, as will be seen in the sequel. I have intimated by notes of interrogation my dissent from those portions of the extracts to which I take exception.

Doctor Markham devotes several pages of his work\* to the discussion of the murmur of roughened or constricted mitral orifice, the quality and significance of which he correctly apprehends, but he falls into the error which Skoda and so many others had committed, of describing it as "diastolic" in rhythm, a mistake of such cardinal importance as to vitiate his entire subsequent discussion of the subject, by rendering his description useless or misleading in clinical study. Whilst admitting with Dr. Markham that systolic mitral murmur is, not unfrequently, associated with presystolic, I cannot agree with him in the opinion that such is the case in most instances, much less that it is invariably so where the constriction is considerable. Neither does my experience accord with the statement that this murmur is frequently audible over the entire precordium, or that it is associated with marked dyspnoea even in a large per-centage of cases, save at a very advanced stage of the disease, when venous congestion, anasarca, and hæmoptysis proclaim pulmonary engorgement and extravasation, and failure of the right side of the heart.

I have met with several examples of presystolic murmur which extended backwards over the entire long pause quite up to the second sound, but in no instance has it extended over the

\* *Diseases of the Heart, their Pathology, Diagnosis, and Treatment*, 1856, p. 206 et sequent.; and *Monthly Journal of Medical Science*, 1854.

period of the second sound, short pause, and a portion of the systole, by which in this connexion I understand the first sound, as alleged by Skoda and Markham.

Nevertheless I freely concede to Dr. Markham the merit of having been one of the first\* to distinguish this from other mitral murmurs, and to recognize and insist upon its special diagnostic value, even though he failed to convey to his readers a correct notion of its rhythm, owing, no doubt, to the laxity of expression in describing the movements and rhythm of the heart permitted at the date at which he wrote.

Doctor Gairdner was undoubtedly in these countries the writer who first gave a clear and full exposition of this subject. In a remarkable paper† he not only lays down rules for the positive identification of this murmur, under the title of "auricular systolic," but contrasts it, chiefly, and very properly, on the ground of its peculiar rhythm, with all other cardiac murmurs whatever.

In his *Clinical Medicine* published the following year Dr. Gairdner reiterates the views which he had previously expressed, and gives‡ the particulars of eight cases of mitral obstruction, with three autopsies all confirmatory of the diagnosis.

Doctor Andrew, writing of presystolic murmur three years subsequently, makes the following statement:§ "So that it is safer to determine the period of the heart's action at which a murmur of this *doubtful* character takes place by its relation to the first sound, than, as is almost instinctively done, by its relation to the impulse. At the same time it must be confessed that the diagnosis of a presystolic from a systolic ventricular murmur *is one of the most difficult tasks in the physical examination of the heart, and is often all but impossible.*|| If the murmur masks

\* In the *Edinburgh Monthly Journal* for January 1854, Dr. Markham gives a very good account of this murmur, under the name of "diastolic mitral murmur," with illustrative cases, in which the principal features of obstruction at the mitral orifice are clearly set forth; he describes the murmur as immediately preceding the impulse of the heart, and as being indicative of mitral obstruction.

† "A Short Account of Cardiac Murmurs," *Edinburgh Med. Journal*, vol. vii., Nov. 1861.

‡ Page 599.

§ *Bartholomew's Hospital Reports*, vol. i., October, 1865, p. 33.

|| The italics are not in the original.

and is continued through the first sound it is pretty surely systolic; if, on the other hand, the first sound is heard of natural quality, and following the murmur, it is with equal certainty presystolic and onward; but unfortunately for the auscultator the changes which occur in the auriculo-ventricular orifice or its neighbourhood, leading to the production of an onward murmur, are very frequently such as to interfere more or less with the ordinary action of the valves, and so far to alter the quality of the first sound as to render the determination of its exact commencement and duration extremely difficult."

Thus, whilst admitting in theory the distinct identity and the significance of presystolic murmur, Dr. Andrew, at the date of his memoir, regarded it as "doubtful," by reason of the difficulty, amounting almost to impossibility, of identifying it in practice.

In some lectures which I published in 1866\* I insisted upon the special diagnostic value of presystolic murmur, and adduced seven cases in illustration, in three of which *post mortem* examination revealed contracted mitral orifice, confirming the diagnosis in two; in the third of these cases, which was not seen till the patient was moribund, no murmur was audible and no diagnosis was made.

Since that date I have paid special attention to this subject, and have met with fifty-six additional examples of presystolic murmur; in all these cases with one exception the diagnosis of more or less obstruction of the mitral opening was confidently made, mainly from this sign, conjoined, however, in several instances with many or all of the special symptoms which characterize the affection, to be mentioned farther on. My cases amount, therefore, to sixty-three in all. In thirteen instances out of this number *post mortem* examination of the body was made, and in all thirteen the mitral orifice was found in a greater or less degree contracted. Two of the thirteen patients who were the subjects of *post mortem* examination were moribund when they came under my notice (*vide* cases of Mrs. Quinn and Mary Armstrong) and owing to the absence of presystolic murmur the diagnosis of mitral constriction was not

\* "Clinical Lectures on Diseases of the Heart," *Medical Press and Circular*, July, 1866.

made. In the remaining eleven a positive diagnosis was made, and was verified by dissection.

Friedreich in 1867 admitted in a qualified sense the diagnostic value of presystolic murmur, declaring that the murmur associated with mitral narrowing is "sometimes" of this rhythm only. He, however, assigns to this murmur ordinarily a "diastolic" rhythm and fremitus.

In 1868 Dr. Sutton\* detailed the particulars of an example of mitral narrowing which had come under his notice, and was announced by harsh presystolic murmur, which, as frequently happens, became inaudible on the occurrence of extreme debility consequent on an intercurrent seizure, in this case vomiting, and became again audible when the vomiting ceased and the patient recovered strength. It finally ceased, however, some time before death, as is the case in the majority of instances. Dr. Sutton adds that within a period of two years twelve examples of presystolic murmur had come under his notice.

Oppolzer one year later still described the murmur of mitral stenosis as "diastolic."† And in the same year Trousseau,‡ speaking of a case of cardiac disease then under observation in one of his wards, said, "Upon auscultation the cardiac lesion was revealed by a double bellows murmur, having its maximum intensity in the situation of the apex of the heart, the blowing accompanying the first sound of the heart was harsh, and that *accompanying the second* was softer. These stethoscopic phenomena were characteristic signs of valvular insufficiency, and of *constriction* of the left auriculo-ventricular orifice."§ In the foregoing extract the illustrious author of the *Clinical Medicine* distinctly intimates his opinion in regard to the "diastolic" rhythm of the murmur of mitral stenosis. In the early part of 1868 also Dr. Henry Simpson of Manchester published|| the substance of a paper on this subject read by him before the Medical Society of that town, in which he not only fully recognized the pathognomonic significance of presystolic murmur,

\* *London Hospital Reports*, vol. iv., 1867-8.

† I quote from Dr. Hilton Fagge's paper already referred to.

‡ *Clinical Medicine*, 1868, Syd. Society's edition, vol. iii., p. 396.

§ The italics are not in the original.

|| *British Medical Journal*, May 16th, 1868.



but likewise adduced in illustration of its diagnostic value five cases, in four of which the diagnosis of mitral narrowing was made from this sign mainly, and in two was confirmed by autopsy. In the first case death occurred suddenly and before a diagnosis had been made.

Dr. A. T. H. Waters, in his work\* wrote as follows on this subject: "But when the mitral orifice is much contracted it is quite conceivable that the auricular contraction may cause a murmur, which, of course, is diastolic as to time, or as some prefer to say, presystolic.

"When there is a double murmur, or even only a diastolic one, at the base, and a double murmur at the apex and towards the left axilla, although the second murmur heard at the apex and to the left may be, and probably is, of mitral origin, still such a conclusion is open to the objection that it may be the aortic diastolic murmur conveyed to the left."

The admission, contained in the last paragraph, of the possibility of confounding aortic diastolic with presystolic murmur, suffices to show that Dr. Waters had failed to apprehend the rhythm of the latter murmur, upon which exclusively its diagnosis depends.

In 1869† Dr. Hyde Salter published the details of six cases, in which a presystolic murmur had been heard, and the diagnosis of mitral narrowing made therefrom. In two of these cases the diagnosis was confirmed by examination of the body after death. In this communication he adverts to the frequency with which the presystolic murmur is terminated by a sharp and clear first sound, an observation the truth of which I can fully confirm. In some of his cases the murmur commenced immediately or shortly after the second sound, and occupied nearly the entire long pause. In reference to this peculiarity of rhythm he inquires "Does the inability of the auricle to empty itself forward provoke it to anticipate its systolic action?" The answer to this question requires that the precise state of the auricle at the several periods of the cardiac

\* *Diseases of the Chest*, 1868, p. 363-4. In the second edition of this work (1873, p. 374) Dr. Waters fully recognizes presystolic or "mitral diastolic" murmur.

† *Lancet*, October 23rd and 30th, 1869.

cycle shall be, in the first instance, briefly reviewed. At the commencement of ventricular systole the efflux from the auricles is abruptly arrested by the closure of the auriculo-ventricular valves, but influx from the cavæ and pulmonary veins continuing, the auricles are progressively but rapidly filled and ultimately distended, a condition to which backward pressure upon the valves from the ventricles in some degree contributes. At the moment of relaxation of the ventricles the auriculo-ventricular valves are suddenly and forcibly thrown open by the removal of pressure from their ventricular surface, and the simultaneous elastic recoil of the walls of the auricles. This, I would call the passive or reactive movement of the auricles, and although due to the elastic reaction of their walls evoked by distension, it is of a forcible and energetic character, and where extreme auriculo-ventricular narrowing exists, capable of developing a murmur which must necessarily be diastolic or postdiastolic in time.

The auricles having thus partially disengorged themselves, and been thereby relieved from the reaction of distension, contract feebly and vermicularly in the direction of the ventricles, as I have repeatedly seen in experiments upon dogs, and likewise in the person of M. Groux through the thin covering of his fissured sternum. At this period likewise, which corresponds with that of the entire long pause less the presystole, a murmur may be developed in cases of extreme contraction of the auriculo-ventricular orifice, and if so developed it should be post-diastolic, or posterior to the second sound, but connected with it by continuity, and yet not strictly presystolic because anterior in time to the presystole. Murmurs of indubitable mitral origin, and corresponding in time to the latter of these periods, I have met with in a few instances (*vide* cases of Anne Coats and Thomas Doyle), and where presumably no organic lesion save that of extreme mitral stenosis existed.\* I believe that herein lies the answer to Dr. Salter's query.

\* I have not met with an example of *diastolic* apex murmur in the strict sense, but Dr. Stokes has mentioned to me a case, and kindly invited me to examine it, (unfortunately I missed the opportunity of doing so), then under his care in the Meath Hospital, in which there were two murmurs, a systolic and a diastolic, at the apex, and both in Dr. Stokes' opinion purely mitral in origin.

I can, at the same time, conceive that a muscular chamber which had not been able to empty itself during the ordinary period allowed for its contraction, owing to narrowing of the orifice of exit, would anticipate the normal period of its next contraction, in proportion to the quantity of liquid remaining in it from the contraction immediately preceding, bearing in mind that the *stimulus* consists in distension. But as a matter of fact does the auricle in such case fail to empty itself during its contraction? Certainly not, so long as hypertrophy of its walls continues to compensate for the difficulty in advance; but ultimately, when the auricles begin to fail in this supplementary function owing to impairment of nutrition and degeneration of tissue, incomplete evacuation and consecutive premature distension must necessarily ensue. It is moreover noticeable, that where the murmur extends far backwards into the long pause the initial portion of it is faint, and that it gradually swells into a loud and harsh sound, attaining its maximum intensity immediately prior to ventricular systole (*vide* case of Thomas Doyle). This peculiarity of the murmur is more consonant with the hypothesis above stated than with that of premature active systole of the auricle.

In explanation of this phenomenon Dr. Hilton Fagge propounds the ingenious hypothesis, that owing to the tardiness with which the left ventricle is filled, and the resultant stimulus of distension applied to it, its contraction is postponed, and the whole series of events dependent upon, and consecutive to it, pushed back in a proportionate degree. Hence the second sound, in the cycle of events, is brought close up to the presystolic murmur, and the retro-elongation of the murmur is only apparent. But it may be objected that the murmur is actually and audibly prolonged in the cases contemplated, and without derangement of synchronism in the movements of the two sides of the heart. I am of opinion, as already stated, that the unusual length of the murmur in such cases is due to a *prefix* not to a *suffix*, and that the former is the result of abnormal effectiveness in the early or undulatory contraction of the auricle, derived from the hypertrophy of its walls.

Doctor Gee\* makes some very judicious remarks on this subject, having reference, however, rather to concomitant phenomena than to the murmur itself. He very truly states that the murmur of mitral constriction is but rarely audible at the angle of the left scapula, and that accentuated pulmonary second sound fails on the establishment of tricuspid regurgitation, because then the contractile energy of the right ventricle is in some degree expended upon the reflux current. I regret to find, however, that Dr. Gee makes use of the term "diastolic" in specifying the rhythm of mitral obstructive murmur, because such laxity of expression tends to embarrass the student, and to encourage scepticism, and is scarcely excusable in an author who professes to write up this subject to the present date.

In 1870 also Dr. Paul Niemeyer† laid down as diagnostic of mitral contraction, "disordered rhythm, systolic, long, loud murmur over the apex of the heart, strong fremitus cataire, and in rare cases also short diastolic murmur." He elsewhere describes this murmur as "diastolic" in rhythm, adding that mitral stenosis is always accompanied with mitral regurgitation. From the preceding it may be concluded that in the opinion of this eminent authority the murmur of mitral stenosis is *diastolic* in time, and that the systolic murmur which he associates with it as a constant accompaniment, is that of mitral reflux, a complication of by no means constant occurrence.

In the early part of 1871 Dr. Hilton Fagge contributed a memoir "On the Murmur attendant upon Mitral Contraction."‡ To this valuable paper, which contains the fullest and best *résumé* of the subject hitherto published, I have already referred. Irrespective of its value as a historic repertory, Dr. Fagge's memoir contains much original matter which entitles it to notice here.

The cases in which a presystolic murmur was heard and identified were twenty-six in number, and out of this number seven *post mortems* were obtained, all confirmatory of the diag-

\* *Auscultation and Percussion*, September, 1870.

† *Handbuch der Theoretischen und Clinischen, Percussion und Auscultation*, band ii., 1870, quoted by Dr. H. Fagge, *loco citat.*

‡ *Guy's Hospital Reports*, third series, vol. xvi., March, 1871.



nosis of mitral contraction. Forty cases are likewise given in which the mitral orifice was found contracted after death, but in which, during life, this condition was not diagnosed. This category is composed of cases in which either no physical examination of the chest, or an imperfect examination was made during the patient's lifetime; secondly, those in which a systolic bruit at the apex was noted; and thirdly, those in which no bruit whatever was detected, the patients being at the time moribund.

The first-mentioned group, or those in which the examination of the patient was inconclusive, are twenty-four in number. These are of no value whatever, and should therefore be set aside, in considering the diagnostic significance and the constancy of presystolic murmur as a sign of mitral narrowing.

The second group (excluding one in which the bruit was diagnosed as that of tricuspid reflux) includes ten cases. Some of these Dr. Fagge had himself never examined, and in at least two others he could not detect murmur of any kind. The majority are quoted from the practice of his colleagues; and considering the meagre and imperfect manner in which they are reported, I cannot attach much value to them as evidence of a want of uniformity in the acoustic sign of mitral contraction. Indeed, Dr. Fagge seems to be of the same opinion, for he does not in his summary dwell much upon them.

The cases, four in number, in which the patients were examined only *in articulo mortis*, and no murmur was heard, serve only to illustrate a phenomenon of constant occurrence, namely, the cessation or complete masking of presystolic murmur for a period varying from several hours to as many days preceding the patient's death, according to the time at which the cardiac asthenia of dissolution has set in.

Dr. Fagge justly observes: "Hitherto, I believe, no single instance has been recorded in which a presystolic murmur has existed during life, and in which the mitral orifice has not been found after death to be very decidedly narrower than usual. It has been supposed by some writers that a mass of vegetation growing from the auricular surface of the valve might obstruct the flow of blood sufficiently to give rise to such a murmur (pre-

systolic). This may be ; but I have found no recorded instance of it. Even its theoretical possibility is, I think, doubtful ; for I am much inclined to believe that an essential element in the production of the presystolic murmur is the vigorous jet of blood caused by an hypertrophied auricle ; and this of course implies a chronic obstruction, such as no mere mass of vegetation would be likely to produce." He adds: "If a patient be suffering from cardiac dropsy, if the heart be beating rapidly and irregularly, and if perhaps there be in addition a loud systolic murmur, *the non-discovery of a presystolic bruit goes but a very little way to disprove, or even to render improbable, the existence of mitral contraction.*" I cannot subscribe the opinion expressed in the words which I have italicised, at least to its full extent. If a careful examination has been repeated once or oftener, the patient having been composed by rest and the warmth of bed, I do not think it likely the characteristic murmur, which I maintain is absent in veritable mitral contraction only for very brief periods and exceptionally, can be missed. It has repeatedly happened to me to *suspect* mitral narrowing at the first examination, notwithstanding the absence of presystolic murmur, and from the character of the first sound exclusively, the patient being chilled and depressed, and only recently put into bed ; and on the following day to make a positive diagnosis from the existence *then* of a distinctly audible presystolic murmur. I shall have occasion further on to revert to Doctor Fagge's views in relation to other departments of this subject.

Doctor Wilks, at the same date and in the same medium, admitted in its entirety the doctrine which I have above advocated. He says : "A direct mitral bruit, however, may probably occur, not only during the contraction of the auricle, but also during the heart's diastole and pause."\* In this passage it is asserted that the murmur of mitral narrowing may be in rhythm cotemporaneous with the second sound ("heart's diastole") the peridiastole ("pause,") and the presystole ("contraction of the auricle"). And, finally, towards the close of 1871 Dr. George W. Balfour† declared his opinion that presystolic

\* *Guy's Hospital Reports*, third series, vol. xvi., March, 1871.

† *Edinburgh Medical Journal*, November, 1871.

murmur "may be conclusively accepted as a distinctive proof of the existence of a permanent deformity, even though the murmur itself should subsequently disappear, as it frequently does." "The mitral orifice," he says, "is usually diaphragmatic." In regard to rhythm, he says the murmur of mitral stenosis often immediately follows the second sound, being sometimes separated by an interval from the true presystolic murmur, and sometimes including it and running right through the periods of diastole and rest up to the apex beat. A soft diastolic murmur, a pause, and a presystolic murmur in succession are due, he thinks, to great narrowing. In the foregoing passages it is virtually admitted that the rhythm of the murmur of mitral narrowing may be diastolic, postdiastolic, or presystolic; and it is added with truth that the two former varieties, whether single or combined, are indicative of extreme contraction of the mitral orifice. I have, however, some doubt as to the constancy of the alleged connexion between prolonged presystolic murmur and sharp first sound, on the one hand, and a funnel-shaped mitral opening on the other.

The diagnostic value of presystolic murmur as a sign of contracted mitral orifice may be judged from the record of cases in which, mainly from the evidence afforded by it, this condition of the mitral orifice has been diagnosed, and been proved by *post mortem* examination to have existed.

Doctor Fagge gives a list of twenty-eight cases from the following sources: Fauvel, 3; Gairdner, 4; Gull and Wilks, 1; Rees, Gull, and Moxon, 2; Hayden, 3; Peacock, 2; Simpson, 3; Hyde Salter, 2; Sutton, 1; Hilton Fagge, 7; a total of 28; to which I have now to add a supplementary list of 9 cases; in all 37.

There can be, therefore, no longer any doubt that with the precautions against mistakes of identity previously mentioned, this murmur is entitled to rank as pathognomonic of a particular organic lesion of the heart, namely, stenosis of the mitral orifice; and I would add, that no other acoustic sign of itself affords equally certain evidence of structural lesion of the heart.

Why stethoscopists had, previously to 1843, failed to appre-

hend this murmur, and assign to it a special significance, may, I believe, be accounted for by reference to a radical error, not even yet entirely exploded, namely, that of refusing to recognize the possibility of the occurrence of cardiac murmurs unconnected by *causal* association with either sound of the heart. Senac truly remarks: "Il en coute moins de decouvrir la vérité que de dissiper les ténèbres dont on l'a enveloppée.\* The universal acceptance of the traditional classification of cardiac murmurs into "systolic" and "diastolic," and the unanimous refusal of pathologists to admit any outside the domain of the sounds of the heart, led, as a necessary consequence, to a forced interpretation of murmurs occurring within the periods of silence. Hence it was that murmurs associated only by proximity with the sounds of the heart were designated as systolic and diastolic respectively.

If it had been realized that both the systole and diastole of the ventricles, and the diastole of the auricles, are continued through the periods of silence, and that the systole of the auricles occurs *only* within one of these periods, the error of assigning murmurs to the periods of sound exclusively would not have been committed. That the direct mitral murmur has been frequently, and by able and acute observers, recognized and described as diastolic in rhythm, the preceding references and quotations will have shown. This error seems difficult to comprehend, owing to the much closer relationship which the presystolic murmur holds to the first than to the second sound. In some instances, I doubt not, the mistake has been made, of assuming the first sound to be the second, owing to its remarkably sharp, clear, and clicking character,† whilst the veritable second sound was dull, and as frequently happens in these cases, partially masked at the apex, so as to resemble rather an imperfectly pronounced first sound.

In other examples, fewer in number, where the two sounds of the heart were correctly identified, I believe the error has arisen

\* *Traité de la Structure du Cœur, de son Action, et de ses Maladies*, 1749, Preface, p. xxix.

† Markham, Gairdner, and Sanders have adverted to this character of the first sound in connexion with mitral stenosis.



from the fact that the murmur occupied the greater portion of the long pause, and was therefore notably out of time with the first sound; in such cases it has been described as "diastolic," in the sense of being non-systolic. The former of these errors, namely, that as to identification of the sounds, may be avoided by applying the finger to the carotid artery whilst the sounds are being scrutinized; it will thus be found that the sound succeeding the murmur is synchronous, or approximately so, with the carotid pulse, and that it must therefore be the first, notwithstanding its unnatural character. The establishment of this fact will at once lead to a correct designation and interpretation of the murmur. The second-mentioned error cannot occur if the auscultator in the first instance recognize the possibility of murmurs within the periods of silence, and then correctly apprehend the rhythm of that under examination. The "binaural stethoscope" of Dr. Scott Alison has been recommended by Dr. Gairdner as an aid to the identification of the sounds of the heart in doubtful cases, one of the bell-ends being placed over the apex, and the other over the base of the heart. I have tried this instrument, but owing to the difficulty of keeping it steadily fixed in the ears, and the rustling noise which it consequently produced, together with the irritation to the ears thence arising, I was forced to give it up after a few trials. The "bruit de rappel" of Bouillaud, or reduplicated second sound, is, no doubt, eminently suggestive of narrowed mitral opening, because associated oftener with that than with any other condition; but it is by no means pathognomonic of this lesion. Further on I shall adduce an example (Case 86, J. Hutchinson) of this phenomenon in connexion with double aortic murmur, where mitral narrowing did not exist. (See pages 127 and 166.)

The identification of presystolic murmur depends mainly upon its rhythm, which coincides with the terminal portion of the long or diastolic pause. It commences at an interval after the second sound, usually considerable, and corresponding to at least two-thirds of the entire pause, but variable in different cases according to the degree of narrowing of the orifice; the greater that degree the more protracted will be the murmur,

and the shorter the antecedent silence by which it is separated from the preceding second sound. A distinctly appreciable interval does, however, exist in every typical case between the second sound and the murmur, whilst in no case does a period, however short, intervene between the murmur and the succeeding first sound.

The rhythm of this murmur is, therefore, accurately described as *presystolic*, a term borrowed from Gendrin, by which is implied that the murmur distinguished by it is immediately antecedent to the first sound, running quite up to, but not infringing upon that sound, and of variable length in the direction backwards, in proportion to the degree of contraction of the mitral orifice, but leaving, with rare exceptions, an interval between it and the preceding second sound.

The murmur is thus more closely related to the first than to the second sound, but in point of fact is distinct from both, and belongs exclusively to the period of normal diastolic silence.

The exceptions alluded to in the preceding paragraph render it necessary, in order to present a synoptic sketch of this murmur under its various modifications, to classify the latter under four heads, as follows:

1. The typical presystolic murmur, which answers to the preceding description, and is characterized mainly by being *prefixed* to the first sound, and separated from the preceding second sound by a long interval of silence.
2. Next in frequency of occurrence to the preceding, often succeeding to it, and representing a greater degree of contraction of the mitral orifice, is a murmur of double rhythm, or broken up into two fragments, one of which adheres as a prefix to the first sound, and represents the ordinary presystolic murmur, whilst the other succeeds the second sound, being appended to it as a *suffix*; these two fragments being separated by a brief period of silence (see Cases of M. E. Coates, M. Ferguson, and T. Doyle).
3. A protracted murmur extending backwards from the first quite to the second sound, covering the whole period of

the long pause, and representing a further development of the preceding disintegrated murmur by a bridging over of the interval of silence, and union of the two fragments, and indicating a still further degree of contraction of the orifice (Cases of T. Doyle, Jane Conroy, and Mary Brennan).

4. A murmur extending not backwards but forwards, and so completely fused into the first sound that it seems to be but the emphasized initial portion of it. This form indicates a much contracted and very rough state of the orifice (Case of Thomas Ward).

The preceding category exhibits concisely the various forms of presystolic murmur which have come under my notice. The three first are set forth in the order of their frequency, and represent, as I believe, so many stages of progressive development of one common lesion. It does not follow that every case must pass through these several stages, and exhibit in its progress the various modifications of murmur above mentioned. Death may result from various complications, or from the effects of the disease itself, in any one of these stages. The last-mentioned form is the rarest of all, and indicates, in an especial manner, a scabrous condition of the orifice, with, probably, a pendulous flap or tongue depending into it, and capable of protracting an ordinary presystolic murmur by vibration.

The transition of the first form into the second, and of the second into the third, in the progress of morbid change of the mitral orifice, may, in my view, be explained by reference to the peculiar rhythm of auricular movement, as already explained at page 206. When obstruction exists only in a moderate degree, the resultant murmur coincides with auricular systole properly so called, or with what I have designated "the momentum of auricular contraction," and therefore immediately precedes the first sound of the heart. When, in the further progress of organic change, obstruction has increased, another murmur is developed, corresponding in time to the period of *passive* influx of blood from the auricle, the result of the passive reaction of its walls from a state of temporary distension, on the cessation of ventricular systole. Hence, this latter murmur is

*postdiastolic* in rhythm, that is to say, it immediately follows the second sound. The interval of silence by which these two murmurs, or rather fragments of the same murmur, are separated, corresponds to the period of normal repose of the auricle, succeeding the passive, and preceding the active contraction of its walls. But where mitral obstruction has advanced to the last degree compatible with the maintenance of a vitalizing circulation, even this interval is bridged over by a prolongation of the postdiastolic element of murmur into the presystolic. Hence the prolonged murmur covering the entire of the long pause indicates the highest degree of mitral obstruction compatible with life.\*

The *quality* of presystolic murmur is always more or less harsh, and usually decidedly so;† it is, in its typical form, much better represented by the *bruit de rouet r r r r* of Bouillaud, than by the *bruit d'aspiration*, which he regards as pathognomonic of contracted orifice.

The harsh and disagreeable quality of the murmur is further illustrated by the frequency with which it is associated with fremitus.

I have elsewhere‡ stated that "a murmur of this character and rhythm, loudest at the left apex, not audible, or faintly so, at the base, and not transmitted, save occasionally to the left side of the lower dorsal spine, may be regarded as diagnostic of mitral obstruction."

A longer experience has afforded me no reason to alter or modify this statement in any particular.

It would be difficult, bearing in mind these specific characters,

\* The distinction above made, and the grounds upon which it is based, are, I am glad to perceive, virtually implied by Dr. G. W. Balfour in a very able article on this subject, published in the *Edinburgh Medical Journal*, for November, 1871.

† I cannot by any means agree with Hope (*opus citat.* p. 83) in the opinion that "no roughness attends the murmurs from influx from the auricles into the ventricles." See my cases of mitral obstruction, *passim*. Doctor Flint (*A Treatise on the Principles and Practice of Medicine*, third edition, 1868, p. 325) on the contrary, revokes the statement which he made in the two first editions of his book, to the effect that presystolic murmur was soft in tone, avowing the conviction forced upon him by longer experience, that the murmur in question is usually harsh and unpleasant to the ear.

‡ "Clinical Lectures," *Medical Press and Circular*, July, 1866.



to mistake presystolic murmur for any other. Nevertheless this error has been not unfrequently committed; mainly, I believe, from failure to correctly appreciate its peculiar rhythm.

Hitherto I have met with only three sources of error in the identification of this murmur, namely: (*a*) Localized apex attrition murmur of presystolic rhythm, and associated with presystolic fremitus; (*b*) Prolonged bellows murmur confined to the area of the apex, replacing the first sound, and extending quite up to the second sound which was associated with diastolic impulse.

In this last-mentioned group the substitutive systolic apex murmur, covering the period of the first sound and of the short pause, and touching the second sound, might readily have been mistaken for a murmur of presystolic rhythm; and the muffled second sound associated with impulse at the left apex, quite as readily mistaken for the first (see Case of Margaret Connor). The diagnosis, however, was not difficult, and rested upon three considerations, namely, the synchronism of the murmur with systolic impulse and carotid pulsation; secondly, the fact that at the base a second sound was heard free from murmur, which, as the stethoscope was shifted by short stages towards the apex, became gradually audible, whilst the second sound became at first faintly, and then distinctly associated with it as the apex was approached. It was manifest, therefore, that the connexion between the murmur and the second sound was entirely casual. Thirdly; the quality of the murmur which was soft and blowing, also furnished a valuable, although an apparently trivial element of negative diagnosis.

(*c*) Reduplication of the first sound at the apex by resolution into its two elements, the former of which, being dull and masked, and immediately preceding the latter which was sharp and might readily be mistaken for the modified first sound of mitral contraction, is liable to lead to a mistake of identity for presystolic murmur. The differential diagnosis, however, may be readily made by reference to the *time* of carotid pulsation, which will be found to *coincide* with the *quasi* murmur; and to the quality of the sound, which is entirely devoid of harshness, a condition, as far as I know, never associated with the murmur of mitral stenosis.

I have already stated that the murmur of mitral obstruction is harsh and unpleasant to the ear; I have never met with an example which constituted an exception to this statement, notwithstanding that Markham maintains that softness is a quality essentially characteristic of presystolic murmur.

The differential diagnosis of apex-friction sound of single and presystolic rhythm is attended with much greater difficulty, because it is usually associated with fremitus of corresponding rhythm, is loud, not transmitted, and not associated with left ventricular hypertrophy. It is, however, not steady or uniform as to rhythm, quality, or loudness; is intensified by pressure, which likewise causes some degree of pain, and is associated with a history of pericarditis, and usually with double frottement if observed early.\* Dr. Chambers' test will also be found to afford assistance in a difficulty of this kind.†

Possibly, also, a murmur caused by patency of the foramen ovale, or by an aneurism of either auricle, may be mistaken for it; I have not, however, met with an example of error in diagnosis arising from either of these causes.

The principle of intersonal murmurs being thus admitted in the general recognition of the typical representative of this class, the presystolic murmur, I fail to perceive on what grounds the remaining members of the group can be logically ignored. Of *postsystolic* murmur I have met with and noted five examples. In three of these the murmur was confined to the area of the apex, and in two out of this number it was non-organic as to cause, traceable in one to excessive tobacco-smoking (case of W. Doyle,) and in the other, a female, to nervousness, and

\* In many cases of pericarditis, limited to the apex, the friction-sound is single and systolic, or pseudo-presystolic from its commencement to its extinction. A careful scrutiny of the rhythm of such, and their subsequent and early cessation, will enable the auscultator to avoid error.

† *Clinical Lectures*. If the ear be slowly and partially withdrawn from the stethoscope applied to the chest at the seat of murmur, the latter will cease to be heard if it be exocardial; whereas, if endocardial, it will be distinctly heard so long as the ear touches the stethoscope, however lightly. I have repeatedly made use of this test in doubtful cases, and have invariably found the evidence afforded by it, whether positive or negative, to coincide with that derived from more trusted sources.

associated with nervous palpitation (Case of "a woman").\* In the third case in this category (that of Catherine Hughes) there was organic alteration of the mitral valve and concomitant hemiplegia.

In one case only (No. 95, Lawrence Carolin) the postsystolic murmur was basic, and due to trivial alteration of the aortic valve, as shown by *post mortem* examination of the heart.

The fifth example alluded to above (case of Catherine Field) was one of pseudo or attrition-murmur, of single rhythm, and by its want of fixity of character, perceptible though slight variation of rhythm, and manifestly superficial location, readily distinguished from organic murmur of endocardial origin.

As to the rhythm of postsystolic murmur, it occupies that portion of the short or systolic pause immediately succeeding the first sound, the *périsystole* of Gendrin. It is always in continuity with the first sound, occasionally coinceptive with it, but extending beyond it into the brief period of silence intervening between the first and the second sound. The pre-systolic and the postsystolic murmur may be therefore regarded respectively as a *prefix*, and a *suffix* of the first sound. In quality likewise the two murmurs present a striking contrast; for whilst, as already stated, the presystolic murmur is invariably harsh and vibratory, the postsystolic murmur has been, within the range of my experience, no less uniformly soft and blowing in character.

Postsystolic murmur is by no means of equal value as evidence of valvular lesion when located at the apex and at the base of the heart respectively. In the former situation it is in most instances due to atony or temporary relaxation of the walls of the left ventricle, in consequence of which, and independently of any degree of structural alteration of the valves, as I have elsewhere explained,† the parietes of the ventricle expand at one or more points under the centrifugal pressure of the contained blood at the acme of systole, and if these points happen to coincide with the attachment of the papillary

\* *Vide* "Neuroses."

† "On the Pathology and Diagnosis of Non-Organic Mitral Regurgitant Murmurs," *British Medical Journal*, December, 1867.

muscles, one or both segments of the mitral valve are of necessity partially lifted off the orifice, and a slight reflux current, commencing with the acme of systole, and therefore with, or immediately consecutive to, the impulse and the first sound, with a corresponding murmur, are the immediate result.

It is conceivable, however, that murmur of this rhythm may be produced at the mitral orifice by incipient or partial valvular disorganization, as yet not sufficiently advanced to cause systolic reflux. I have not however, met with an example which would warrant a more positive statement upon this subject.

At the aortic orifice, on the contrary, so far as I have observed, postsystolic murmur is invariably caused by valve-lesion of an obstructive character, but partial in degree, and quite inadequate to the production of valvular incompetency. In the only example which has fallen under my notice I incline to think the murmur, though postsystolic, was likewise systolic in time; in other words, that it commenced *with* the first sound but extended beyond it into the short pause; the former portion being masked by the normal sound.

I have met with only one example of *prediastolic* murmur, and in that I was afforded the advantage of a *post mortem* examination. The murmur was aortic in site, and one of the segments of the aortic valve was thick and perforated, and when closed, occupied a lower level than the other two segments (see case of Delany, disease of aortic valves.)

A pericardiac friction-sound may have this peculiar rhythm, at least seemingly, or even actually for a brief period, as illustrated by the case of P. Costello.\*

In reference to the phenomena of precordial morbid sounds, pericarditis holds a position analogous to that which hysteria occupies in relation to subjective manifestations; it mimics or simulates each of them in turn, yet in such fashion that it is with no great difficulty distinguishable from them.

*Postdiastolic* murmur is a veritable sign of much significance and not rare occurrence. As its name implies, it immediately follows the second sound, seeming to arise out of it, and extend-

\* This murmur is not included in the scheme at page 187, because when that scheme was drawn up I had not met with the above, or any other examples of murmur of this rhythm.



ing beyond it to a variable length into the long pause. It is, therefore, the opposite of the presystolic murmur, which commences towards the close of the long pause, and extends up to the first sound.

Of murmur of this rhythm I have observed eleven examples; four were located at the apex, and seven at the base of the heart; the former were harsh, the latter soft in quality.

Out of the latter category I was afforded two *post mortem* examinations of the heart. In both these cases the murmur was aortic, and superadded to a sharp and clear second sound, and in both the aortic valves were incompetent. In one of these (Case 78, John Lalor) which yielded also a systolic basic murmur, the sigmoid valves of the aorta were rolled or shrivelled up at their free margins, so as to oppose a seam-like barrier to efflux from the ventricle, and likewise to admit, by incompetence to close the orifice, a reflux axial current during diastole.

In the second of these cases (Case 28, William H.) there was no murmur whatever save the postdiastolic, which was basic in origin; and dissection revealed a state of the aortic valves incapable of opposing an obstacle to the outflowing current, but incompetent to completely close the orifice and prevent reflux; thus, the antero-right segment was opaque and somewhat thickened throughout the "lunula," and drooped by this portion towards the ventricle, somewhat below the level of the other segments, so as to allow partial regurgitation under high pressure.

In both these cases the sigmoid valves, though incompetent to close the axial portion of the passage, were otherwise structurally healthy, and capable of yielding a sound of tension.

I am warranted, therefore, in regarding postdiastolic murmur as pre-eminently basic in location, and affording evidence, when so situate, of disorganization of the sigmoid valves engaged, by no means advanced, and permitting only partial reflux at the acme of arterial reaction, without entailing loss or even impairment of the second sound of the heart.

This murmur, at the apex, is theoretically important. In the only four cases of it so located which I have met with, there

was undoubted organic disease of the heart. In one of these (Case of Mary E. Coates) I have made the presumptive diagnosis of aggravated mitral constriction, a condition which may be readily conceived competent to give rise to a murmur of this rhythm, by causing an eddy in the current entering the ventricle by the mitral orifice at the first moment of diastole.\* At this moment, as previously shown, the blood which has been accumulating within the auricle during ventricular systole, is urged forward into the ventricle by the pressure of accumulation *a tergo*. If the constriction of the orifice happen to be very considerable, a murmur may be produced by this influx, and coinciding in time with the initial portion of the long pause, the *péridiastole* of Gendrin. The murmur so produced would be not inappropriately designated an apex-postdiastolic murmur.

It will be perceived, however, that I ventured on only a *presumptive* diagnosis in this case, firstly, because I had then met with only one other example, to be presently mentioned, of murmur of this rhythm at the apex; and secondly, because in neither had I the advantage of the conclusive evidence afforded by an autopsy.†

In the second example of apex postdiastolic murmur previously mentioned (Case of Eliza Donaghan) I have reason to conclude that the murmur was of a *quasi* nature, and due to pericarditis, illustrating the multiform or protean character of the acoustic signs of this disease.‡

\* Since the above was written (1870) this patient has been repeatedly examined by me, the physical signs remaining unaltered. In March, 1873, she was again admitted into hospital for dyspnoea and hæmoptysis. The murmur was then of double rhythm, *i.e.*, it consisted of a postdiastolic and a presystolic element. She is still (June 22, 1873) in hospital, expectorating large quantities of blood from time to time, and passing it alternately from the vagina, and the murmur remains as at the date of her admittance (see case).

† I have, since the above was written, met with two other examples of murmur of this rhythm at the apex, and included them in the preceding total. The “presumptive” diagnosis of that date has since become *positive*, as already stated, and the sign itself, in place of being merely suggestive, has assumed in my mind a pathognomonic significance.

‡ I do not include in this category examples of presystolic murmur prolonged backwards into the *péridiastole*, or in other words, of postdiastolic and presystolic murmurs conjoined. Of such the case of the boy Thomas Doyle presents a good example.

The designations *systolic* and *diastolic* as applied to murmurs, although meaning strictly coincidence in time with ventricular systole and ventricular diastole respectively, and therefore including the pauses as well as the periods of sound; have come by conventional usage to imply synchronism with the sounds only.

No doubt this limitation was due to the physiological error to which Laennec gave currency, namely, that the sounds of the heart represented the periods and the duration of its state of activity, and the pause or silence a state of complete repose of the heart. I have already endeavoured to show that there is no repose of the heart in the sense of total absence of movement, active or passive, of its chambers: whilst the ventricles are in a state of active contraction the auricles are undergoing passive expansion, and *vice versa*.

Now, although passive expansion or relaxation implies diminished disintegration of tissue, and nutritive renovation, it by no means implies absence of movement; and whilst one set of chambers is in this state of *passive* movement, the other set is in a state of *active* movement of corresponding duration. If this view be correct, and I believe it will not now be questioned, there is absolutely no period of the cardiac cycle during which two of its chambers are not in active contraction, and two others undergoing passive expansion.

The terms *systolic* and *diastolic*, although therefore critically incorrect in their usual application, as distinctive of the special rhythm of cardiac murmurs, have come, nevertheless, to acquire a definite meaning and value in pathology, and therefore may be retained with much convenience.

The precise *relationship* of murmurs to the sounds of the heart, as well as to the periods of the cardiac cycle occupied by those sounds, should receive careful attention, because of its great diagnostic value, not so much in regard to the nature and the site of the disease, as to its degree and prospective gravity.

In this sense cardiac murmurs may be conveniently classified as *substitutive* or *replacing*, and *concomitant* or *accompanying* murmurs.

The former, as superseding the normal sounds, indicate a greater degree or more advanced stage of valvular disorganization than the latter, which merely accompany them; and although the valves may be utterly disorganized at the outset owing to the extreme violence and gravity of the disease, or to the serious character of the mechanical injury which they may have sustained, and murmur alone have come to represent the normal sounds *ab initio*, the process of disorganization is usually much less rapid, and its progress may be noted with accuracy from month to month, chiefly by reference to the altered relationship of the murmur to the sounds as above indicated. I have repeatedly followed the downward progress of a case in this way from its earliest period to the end.

The pitch and quality of murmurs have been already discussed. The terms made use of by authors to designate these in their several shades remain to be enumerated, together with the causes to which they have been attributed respectively.

1. *Bruit de soufflet* (Laennec) is of high pitch, and caused, according to Laennec, by spasmodic contraction of the heart or arteries; and according to Bouillaud, by vegetations on the valves incompetent to produce obstruction. Hope maintains that this murmur, as all other organic murmurs in their degree, is due to modifications in the current, occasioning increased friction and vibration. I regard regurgitation as *par excellence* the cause of intracardial bellows murmur; and further, I can recall very few examples of obstructive murmur which were, strictly speaking, of this character, and still fewer regurgitant murmurs of the opposite type.

I am bound, therefore, to conclude, that although friction has a share in the causation of reflux murmur, it is not by any means the principal cause of that phenomenon, which, owing to the special character of softness which it all but uniformly presents, I incline to attribute to the condition which is common to all cases of regurgitation, namely, commixture of two currents moving, or tending to move, in opposite directions.

Laennec held that *bruit de soufflet* was the result, as above stated, of simple spasm, and did not indicate the existence of any organic lesion of the heart or arteries. Its rhythm he held



to be almost always diastolic; whereas Bouillaud maintains it is most frequently systolic. With this latter opinion my own experience coincides; it amounts to this, that the number of examples of valvular disease, including *all* stages, in which bruit de soufflet is systolic in time, vastly exceeds that in which it is diastolic. When it is borne in mind that all valvular lesions of inflammatory origin are at the outset proclaimed by murmurs of a soft and aspiratory character, it will only be necessary to add that mitral and aortic systolic murmurs conjointly, exceed in number aortic diastolic murmurs, in order to establish the truth of the foregoing proposition.

Bouillaud likewise asserts that narrowing of one of the orifices is, by giving rise to increased friction, the cause of murmur in ninety-nine out of one hundred cases where it exists. M. Piorry says not once in twenty cases. There is always associated with mitral narrowing, save where great debility of the heart exists, a murmur, but of a harsh character.

Under the generic title of bruit de soufflet, Laennec included four species, namely:—

(a) *B. de soufflet, properly so called.* This is associated by Bouillaud with stenosis and indurated valves arising from fibrous or cartilaginous, rather than from calcareous, change.

(b) *B. de scie*, which is attributed to the same cause, with calcareous induration, by Bouillaud.

(c) *B. de râpe.*

(d) *B. de lime à bois.* The two last are caused by great constriction, with calcareous rough and uneven valves, and strong contraction.

To the preceding Bouillaud adds, as a fifth species of the bellows murmur,

(e) *B. de roucoulement.*

I cannot perceive the slightest resemblance between the *genus* and the alleged *species* above mentioned, with the exception of the first. On the contrary, I regard them as in no respect german, and even as representing opposite pathological conditions; the former, softness of texture, smoothness of surface, and inadequacy of valves; the latter, rigidity or calcification of tissue, roughness of surface, and in most instances obstruction of valves.

2. *B. sec dur parcheminé*, associated with thickening and rigidity of the valves, especially the mitral (Bouillaud).

3. *B. d'aspiration*, indicative of mitral constriction, according to Bouillaud. I have already stated my opinion that this does not represent the murmur of mitral narrowing.

4. *B. de scie*, *ssss* prolonged, (do.).

5. *B. de rouet*, *rrrr* prolonged, (do.).

This last I believe fairly represents the usual quality of pre-systolic murmur, as already stated.

6. *B. de râpe* (do.).

7. *B. de lime* (do.).

8. *B. de sifflement musical* (do.).

9. *B. de fusée* (do.).

Murmurs of the quality of any of the six last mentioned arise from stenosis with indurated valves, according to Bouillaud.

10. *B. de flot* (do.).

11. *B. de miaulement* (do.), in mitral narrowing.

12. *B. de tintement métallique* of Bouillaud, and attributed by him to ossification of the heart or pericardium. I have met with three examples which correspond with the description of this murmur given by Bouillaud, and which, I think, would be more appropriately named *B. de tintement gastrique*, because in my opinion due to gastric resonance imparted to the right ventricle of the heart through the diaphragm. It is not always systolic, as stated by Bouillaud; of three examples of it which have come under my notice, it was postdiastolic in two, and systolic in one only. In two of these it was strictly confined to the sixth and seventh costal cartilages about one inch to the left of the sternum, and in the third to the base of the xyphoid cartilage. The resonance was peculiar and unmistakeably gastric, and consisted rather in an after-tone or echo than an accompanying ring.

13. *B. de timbre enroué, âpre ou fêlé*. (do.), depends upon a tumid, soft, and spongy state of the valves.

14. *B. rauque* (do.). These two last-mentioned murmurs represent endocardial murmurs of the lowest pitch.

15. *B. de frôlement* (do.), indicative of earliest stage of peri-

carditis, in which the serous surfaces are dry, and as yet free from false membrane; but when it is very loud and decided, resembling the bruit de râpe or bruit de scie, there is already false membrane. This murmur resembles the noise made by the rumpling of silk or new paper; it is usually double, more pronounced during systole than during diastole, superficial, as if produced close to the ear, and diffused. In all these particulars it differs from valve-murmur.

16. *B. de cuir neuf* (Collin), also diagnostic of pericarditis, but in the stage of false membrane which has become dense, firm, elastic, partially-adherent, and is subjected to strong tension; it is less common than the last-mentioned, and especially accompanies ventricular systole.

17. *B. de raclement* (Bouillaud), due to grating of rough and hard surfaces, and isochronous with the action of the heart, or of double rhythm; it indicates osteoid plates in the pericardium.

The three last-mentioned *quasi*-murmurs are modifications of the attrition sound of pericarditis, for which the title of "murmur" is highly inappropriate; whenever a veritable bellows murmur is associated with pericarditis Bouillaud regards it, and very correctly in my judgment, as evidence of the complication of endocarditis.

18. *B. de frottement sec* (Gendrin), of systolic and diastolic rhythm, superficial, confined to the precordium, and due to the forcible rubbing of the opposed surfaces of a healthy pericardium upon one another during palpitation.

19. *B. de va-et-vient* (Gendrin), due to inadequacy of the aortic or pulmonic valves, and being in rhythm, according to Gendrin, *périsystolic* and *prédiastolic*. This is the "to and fro" murmur of modern writers, of which the former element is undoubtedly systolic, and the latter diastolic in rhythm.

I have excluded from my classification murmurs of alleged prediastolic rhythm; but, as already stated (p. 220), I have recently met with a veritable example of murmur of this rhythm, and had the advantage of associating it with a definite valvular lesion by *post mortem* examination of the body.

I shall now advert to other and more special characteristics

commonly made use of to distinguish murmurs. But inasmuch as these features imply a foregone conclusion as to the special site and effect of the structural change which has given rise to the murmur, they have not been considered in connexion with those already discussed, which *lead to* rather than *assume* a specific diagnosis.

Of such a character are the designations *mitral*, *aortic*, *tricuspid*, and *pulmonic*; which become still more specific and precise in conjunction with the terms *direct*, *obstructive*, or *afflux*; and *indirect*, *regurgitant*, or *reflux*.

Thus, a substitutive regurgitant mitral murmur would mean the same thing as a substitutive systolic left apex-murmur.

The latter, however, would be the preferable formulà to adopt; because, as already stated, it leads to, rather than anticipates a conclusion.

As previously mentioned, Laennec held that bruit de soufflet was proximately caused by simple spasm.

Gendrin regarded friction as the sole cause, and accordingly named the most simple murmur *bruit de frottement*.

Reflux is, *par excellence*, the cause of bruit de soufflet, as M. Filhos long since urged.\*

Bouillaud considered narrowing of one of the orifices, by increasing the friction between the orifice and the blood, as the principal cause.

According to the same authority, the second cause of B. de soufflet, in the order of numerical frequency, is fibrinous concretions engaging in the orifices.

Third. Hypertrophy and dilatation of the left ventricle consecutive to aortic stenosis.

Fourth. Tumefaction of valves, as in an early stage of endocarditis; vegetations and cartilaginous or calcareous plates upon them without alteration in size of orifice.

Fifth. Adhesion of valves to adjacent walls of ventricle, and consequent inadequacy.

Sixth. Dilatation of any of the orifices, and valvular incompetency arising therefrom.

Seventh. Hypertrophy with dilatation of the left ventricle.

\* *Dissertation Inaugurale.*



Bellows murmur likewise occurs occasionally after fatigue, effort, or moral emotion, in connexion with violent action or palpitation.

Eighth. Chlorosis and anæmia; murmur of a faint character.

Ninth. A temporary bruit de soufflet after great loss of blood.

Bouillaud says that reflux murmur, aortic or mitral, is single, whilst that of constricted orifice is frequently double.

The former proposition is all but universally true; as valvular disease, whether mitral or aortic, *primarily* regurgitant, very rarely assumes in the progress of organic change the character of the twofold lesion. And seeing that, in tissues which are themselves the seat of chronic disease, wasting is the all but universal law, this truth might have been theoretically accepted.

In regard to the second assertion, viz., that the murmur of constricted orifice is frequently double, I find that such was the case in nearly one-third (eighteen out of sixty-three) of my cases of mitral narrowing; whilst, of thirty-three cases of aortic obstruction, the murmur was double in twenty-six.

I have not met with an example of adhesion of the valves to the walls of the ventricle, (No. 5) as a cause of valvular incompetency. Of valvular inadequacy arising from dilatation of the orifice (No. 6) without structural disease of the valves, I have observed and recorded several examples, in which the lesion was either aortic or tricuspid, but very few indeed in which it had its seat at the mitral orifice.

The examples of bellows murmur (No. 7) attributed to fatigue, moral emotion, palpitation, etc., I regard as instances of a dynamic murmur due to atony and temporary yielding of the walls of the left ventricle, with consequent reflux.\*

Laennec says:† “Bruit de soufflet may accompany the diastole of the heart, and that of the arteries, and is so associated with them that it replaces and supersedes entirely their natural sound, so that *at each diastole the ventricle, the auricle, or the artery* in which the phenomenon takes place, yields distinctly a bellows sound which ceases during systole.”

In the words which I have italicized, the illustrious Laennec

\* See Paper by the author, *British Medical Journal*, December, 1867.

† *Traité de l'Auscultation Médiate*, tom. ii., p. 422.

clearly implies his acquaintance no less with murmurs of reflux than with those of afflux; and no less plainly intimates the distinction between systolic and diastolic murmurs, although not admitting them by name, and attaching undue importance to the less effective movement of diastole. Thus, for example, the auricular diastolic murmur of Laennec, coinciding with ventricular systole, would be a systolic murmur in modern terminology, and therefore either mitral regurgitant, or aortic obstructive. The same may be said of his arterial diastolic murmur; whilst his ventricular diastolic murmur would be, according to modern interpretation, either aortic regurgitant, or mitral obstructive; the latter being incorrectly regarded as diastolic.

Notwithstanding the indubitable priority of Laennec in promulgating, at least impliedly, the doctrine of regurgitant murmurs as above shown, Hope claims to have been, in his work published in 1831, the first to describe and invite special attention to murmurs of regurgitation.

No doubt he described them more specifically, and with greater precision than did Laennec; but to assert, as he does, that the French authors prior to himself, including Laennec, were unacquainted with the phenomenon of reflux as a cause of murmur, seems somewhat in excess of what facts warrant.

The *area of apex murmurs* may be represented by a circle of about three inches diameter; or rather by a series of concentric circles or zones, of which the external would possess this diameter, and the central, corresponding to the area of apex pulsation, a diameter of about one inch. The centre of this circle, at which the pulsation of the apex may be detected by the hand placed flat upon the precordium, is located in the fifth intercostal space of the left side, one inch inside the nipple line, two inches from the edge of the sternum, and about the same distance below the nipple-level. The localization of this area depends entirely upon that of the apex itself, which is subject to variation dependent upon (*a*) the position of the body, (*b*) the relative volume of the two ventricles, and (*c*) the volume and condition of adjacent organs. The position of the apex may be ordinarily determined by the application of the hand as already mentioned; this may be accomplished most readily and

satisfactorily by means of that portion of the palm corresponding to the roots of the fingers, which possesses a tactile sensibility of great delicacy in regard to impressions of this kind, due, probably, to the great extent of surface presented, by which the advantage of comparison with the adjacent cutaneous surface is obtained. In the event of no apex pulsation being discoverable, as occasionally happens, for example in great obesity and in the subjects of weak-acting or fatty heart, a slight inclination of the body to the left side, or placing it in the prone position, may enable the examiner to detect the apex-beat. Where even these measures fail, the point of the thoracic surface corresponding to the apex may be easily determined by fixing that of greatest intensity of the first sound, which coincides with the apex-point, and may be quickly ascertained by a practised auscultator. It must be remembered that the position of the apex varies slightly with that of the body, shifting about half an inch to the right or left side with a corresponding inclination of the body, when not opposed by strong and extensive pericardial adhesion, or by increased volume of the subjacent lung. In cases of hypertrophy of the left ventricle with or without dilatation, but especially in the former, the apex is displaced to the left, and may be found pulsating at any point within a range extending two inches outside the nipple line. Or the displacement of the apex may be in the direction downwards and outwards beneath the sixth rib; or even into the sixth intercostal space and outwards to a distance of half an inch to an inch external to the nipple line. The former of these displacements, which are usually due to mitral regurgitation, are the consequence of globular enlargement of the left ventricle; whilst the latter, which, when not caused by displacement of the heart in its totality, are pathognomonic of aortic reflux, indicate elongation as well as increased diameter of the left ventricle. Hypertrophy with dilatation of the right ventricle may alone suffice to cause partial displacement of the apex to the left; but inasmuch as, unassociated with hypertrophy of the left ventricles it is usually dependent upon pulmonary emphysema, the apex-beat is rarely perceptible, being masked by this affection. Being generally preceded and accompanied by left ventricular hyper-

trophy, increase of volume in the right ventricle contributes in a proportionate degree to the left lateral displacement of the apex, which is justly attributed in greatest part to the affection of the left side of the heart.

General pulmonary emphysema has the effect of displacing the heart downwards and inwards, and therefore of abolishing its pulsation in the ordinary site. In such cases the heart may be felt pulsating in the scrobiculus cordis, and its sounds heard most distinctly at the ensiform cartilage. This anomaly is explained by the state of the lungs and the depression of the diaphragm.

The apex of the heart may be displaced to the right or to the left in various degrees by liquid effusion into the opposite pleura; and by cirrhosis or other contraction in the volume of the lung it may be drawn towards the affected side. I have seen the heart displaced upwards and inwards, and pulsating beneath the left clavicle, in a case of inferior thoracic aneurism (see Aneurism, Case of Fay); and I have likewise found it displaced upwards and outwards by a circumscribed purulent accumulation in front of the liver, connected with and derived from an empyema of the right side.

A murmur audible only within the area of the apex, and with greatest intensity at the point of apex-pulsation, would be of mitral origin, whether presystolic, systolic, or postsystolic. Presystolic murmur is, in the majority of cases, strictly confined to the apex-area. Systolic murmur, when of moderate strength, is likewise limited to this space; but a loud murmur of systolic rhythm, and irrespectively of its quality, is transmitted upwards in the left axillary line to an extent proportionate to its intensity, and is likewise audible in the left back near the inferior angle of the scapula. Postsystolic apex-murmur is usually soft and faint, and its diffusion is limited to the area of the apex. The two former are usually, but by no means invariably, associated with accentuated second sound in the pulmonary artery, as first pointed out by Skoda.\* This phenomenon, itself directly dependent upon hypertrophy of the

\* *A Treatise on Auscultation and Percussion*, translated by Markham, 1853, p. 232-3.



right ventricle, will of course be absent in the early stages of both lesions previously to the increase in volume of the right ventricle; and throughout the disease in those cases in which the narrowing of the orifice or the incompetency of the valves exists only in a minor degree, although sufficient to give rise to murmur.

The cause of mitral murmur is usually structural alteration of the valves or of the orifice, competent to oppose in greater or less degree the flow of blood from the auricle into the ventricle, or to prevent the closure of the valves in a degree sufficient to preclude reflux.

This structural alteration is, in the great majority of cases, if not in all, of inflammatory origin, and the direct result of antecedent endocarditis, which manifests a predilection for invasion of the fibrous structures in and about the orifices; it consists in interstitial cell-proliferation and deposit upon the valves and tendinous zone. By this means the valves are thickened, uniformly or at intervals, and not unfrequently attached to one another, or rolled up into a dense mass, offering at once obstruction and rendering closure impossible. In such extreme cases the attached chordæ tendineæ are likewise thickened, and elongated or shortened according to the fixed position of the valves.

When the valves are thus utterly disorganized, not only are their functions annulled in regard to the flux and reflux of the blood-stream, but likewise with respect to the production of tension-sound; hence, in such cases, the systolic or reflux murmur is substitutive; in other words it replaces the first sound, or by depriving it of the valvular element, reduces it to the condition of a simple impulse-sound. In less extreme cases the lesion may consist in a nodular or wart-like deposition or outgrowth on the surface or edge of the valve-segments or tendinous ring. If on the auricular surface and at the attached margin of the valve, such formation may simply interrupt the passage of the blood in its normal course, and give rise to direct, obstructive, or presystolic murmur only; the unaltered lunulæ of the valves being competent to effect occlusion of the orifice in systole, and yield a tension-sound. Hence, in such cases, which constitute only a very small per-centage in obstructive

lesion of the mitral orifice, the first sound is normal and forms a natural pendant to the murmur. In most of these cases, however, the valves are thickened and incapable of yielding sound, which must be due to tension of the tendinous chords. If, on the other hand, the lunulæ have undergone such structural alteration, whether by thickening, excrescence, or shrinking, as to be no longer capable of effectually closing the orifice, but without opposing in any degree the efflux of blood from the auricle, a systolic murmur only, accompanying or substitutive according to the structural condition of the valves, will be the result. An eddy may be produced in the blood-current from the auricle into the ventricle by a minute outgrowth from the auricular surface of the valve, or by a flake of fibrine whipped from the blood and entangled in the meshes of its free margin, and an afflux-murmur of presystolic rhythm be so developed. In such a case, whilst the murmur would afford indubitable evidence of the position of the mechanical impediment, the symptoms, whether subjective or objective, would be null, owing to the absence of any sensible impediment to the circulation; and if the valve-lesion be not progressive, as not infrequently is the case, it may exercise no influence upon the duration of life, and the apparent anomaly would be presented, of a person with organic disease (?) of the heart, as declared by physical evidence, utterly unaware of its existence, suffering no inconvenience of any kind from its presence, and living to the full period even with enjoyment under it. Examples of this kind are comparatively numerous, and afford a striking illustration of the impropriety of announcing openly "disease of the heart,"—a phrase pregnant with the most alarming significance to the uninitiated—in all cases indifferently from physical evidence alone.\* Calcareous degeneration of the valves and adjacent structures whether normal or adventitious, although of more

\* I feel bound, however, to state, that although I have met with several examples of presystolic murmur (*vide cases passim*) of an imperfectly pronounced character, but with the peculiar rhythm, in persons enjoying fairly good health, and even the pleasures and gaieties of society, and have repeatedly examined them, I have no *post mortem* evidence to produce in confirmation of the above doctrine. I adhere to the opinion, therefore, that the characteristic harsh presystolic murmur is pathognomonic of mitral narrowing.

frequent occurrence at the aortic orifice, is occasionally met with at the mitral opening. The subjects of it are usually persons of middle or advanced age and gouty diathesis; the youngest persons in whom I have had demonstrative evidence of its existence were aged twenty-three and twenty-six years respectively (cases of P. MacD. and A. M.). In this form of valvular lesion the endocardial investment of the cretaceous masses has been destroyed; and the subjacent surface, rough by the mode of aggregation of the adventitious particles, is presented directly to the blood, and gives rise to a harsh, filing, or rasping murmur.

As to the pathology of this form of valvular degeneration, I incline to the opinion that it is not necessarily, or even usually, preceded by acute inflammation of the endocardium. In several examples within my personal experience it has been found where there was no history of rheumatism, gout, or acute affection of the heart. In these cases I am forced to regard the valvular lesion either as a primary degeneration of a special structure, and belonging to the same category as atheromatous transformation of the arterial coats; or as the result of degenerative change in the products of chronic inflammation.

The inflammatory deposits in or upon the surface of the valves not unfrequently, in chronic cases, assume a cartilaginous density and elasticity, and a pseudo-cartilaginous structure. The endocardial covering is, however, unbroken, and the surface of the thickened and irregular valve therefore smooth. Hence, the resultant murmur is the reverse of harsh in quality.

An occasional though rare cause of mitral systolic or regurgitant murmur is rupture or disintegration of one or more of the chordæ tendineæ, in consequence of which the corresponding valve-segment is reversed in some degree at the acme of ventricular systole, and partial reflux takes place. The reversion of the valve, unless in the event of detachment of several tendons, an occurrence of extreme rarity, must be incomplete, as each segment receives a group of tendons from both papillary muscles. Irrespectively of the history, I am not acquainted with any specific character either in the symptoms or physical

signs by which lesion of this form may be differentially diagnosed. (See Case 24.)

The anatomical position of the root of the aorta would seem to imply, that the point of maximum intensity of aortic murmurs should coincide with the left third sterno-costal articulation and the adjacent portion of the margin of the sternum; but as the vessel is here overlain by the infundibulum of the right ventricle, the sounds emitted from it, whether normal or morbid, are more distinctly heard somewhat to the right, or at mid-sternum. This point is, however, liable to variation; thus, in pulmonary emphysema engaging the anterior edges of the lungs, the heart is displaced downwards with the diaphragm, and the point of maximum aortic distinctness is correspondingly depressed. Similar displacement results from great hypertrophy of the heart, simply by its weight. Enlargement of the left lobe of the liver may, on the contrary, displace the heart upwards, and elevate the aortic point to a corresponding extent. The transmission of aortic as of other murmurs is governed by the law of current-conduction; hence those of systolic rhythm may be heard, with scarcely diminished intensity, from the mid-point of the sternum upwards along the middle line and right margin of that bone to the level of the first intercostal space, and thence upwards in the course of the great cervical vessels, beneath the right sterno-clavicular articulation, into the right side of the neck; and usually also, but with less distinctness, across the lower portion of the manubrium sterni to its left margin, and upwards into the neck and beneath the clavicle in the course of the left carotid and subclavian arteries. Systolic aortic murmur of extreme loudness and special harshness, if associated with general atheromatous transformation of the arteries, may be heard even in the primary and secondary arterial trunks throughout the body. I have heard such a murmur not only in the descending thoracic and abdominal aorta, but likewise in the brachial, radial, iliac, and femoral arteries. This murmur is also transmitted downwards as far as the ensiform cartilage, but is very faintly audible at that point.

Diastolic aortic murmurs have a much more limited range of diffusion; they are distinctly audible at the xyphoid cartilage,



and over the sternum to the right of the mesian line as high as the second costal cartilage; but rarely, according to my experience, in the aortic arch beyond the first curvature, and never in the carotid or subclavian arteries except when associated with aneurismal dilatation of these vessels.

Doctor Gairdner asserts\* that diastolic aortic murmurs are invariably propagated into the arteries of the neck, "though sometimes very faintly." I have not met with a single example of diastolic aortic murmur which was audible *even faintly* in the carotid or subclavian arteries; whereas, systolic aortic murmurs may invariably be heard in these vessels.

But aortic murmurs, both systolic and diastolic, are occasionally audible at the apex with much distinctness, and in other directions likewise far beyond the limits of the aortic area. I have heard them beneath both clavicles, and even in the left back. In such examples, which are strictly exceptional, the left ventricle is not only hypertrophied but dense and firm in texture, and acts with unwonted vigour; and both it and the osseous framework of the chest serve as conducting media of sound. I cannot conceive a careful observer committing an error of diagnosis in such cases, due attention being given to the point of maximum intensity of the murmurs, and to the increasing faintness with which they are heard as the stethoscope is gradually shifted from this point, as a common centre, to the utmost limits of diffusion. Again, the identity of the systolic aortic murmur may be always crucially determined by its transmission into the carotids, a feature which distinguishes it from all other valvular murmurs. A diastolic murmur at the apex is presumably *not* mitral. I cannot recall from my personal experience a single example of mitral murmur strictly diastolic in time.†

A pericardial friction sound of diastolic rhythm, and closely simulating an endocardial murmur, may, no doubt, be heard at the apex, and even be confined to that situation (case Eliza Doyle); but the identification of this by means of its special characteristics, and above all, its rhythmical variations from day to day,

\* *Edinburgh Medical Journal*, vol. vii., 1861-2, p. 451.

† See foot note, page 206.

cannot be attended with much difficulty. A postdiastolic aortic murmur is not unfrequently heard at the base, to which it is usually limited; it is invariably soft and blowing, and, as its designation implies, it succeeds without interval, or by continuity, a second sound scarcely if at all differing from the normal sound in character and intensity.

I have already expressed my opinion as to the special pathological significance of this murmur; to the effect that it implies a partial, and usually an axial reflux at the orifice of the aorta, the valves being either in a very slight degree altered from the normal state, or structurally sound. I regard this murmur, then, as evidence either of a transitional state of the valves, an early stage in the process of valvular disorganization, or of dilatation of the aorta without valvular disease. It is therefore of much value as an aid to precise diagnosis, and in determining the probable duration of life.

*Tricuspid* murmurs, or those having origin at the right auriculo-ventricular opening, though much less common than murmurs at the mitral orifice, are by no means infrequent. They are usually systolic in rhythm, soft in character, and most distinctly audible to the left of the middle line of the sternum at the level of the sixth costal cartilage, but diffused throughout the tricuspid area, beyond which they are never heard. A presystolic murmur is occasionally developed at the tricuspid orifice; its rhythm is identical with that of mitral origin; it is, however, less loud, as might be inferred from the relative thickness and strength of the two auricles, and its point of greatest intensity corresponds to the fifth intercostal space one inch to the left of the sternum. It indicates narrowing of the tricuspid orifice.

Of this lesion I have met with only three examples (cases of P. McD., Mary M., and Michael F.). In all three, tricuspid was associated with mitral constriction; and in one of these the latter lesion alone was diagnosed, the supplementary diagnosis of tricuspid narrowing was not made, although it was remarked and specially noted that presystolic murmur was distinctly audible near the left margin of the sternum. In the second and third cases the twofold lesion was diagnosed.

Enlightened by my experience of the two former cases, in which the body was examined after death, I would in future regard the existence of two centres of presystolic murmur with or without fremitus, viz., at the apex, and somewhat to the right of that situation, in conjunction with marked systemic venous engorgement, as evidence of the double lesion of mitral and tricuspid stenosis. From these premises the diagnosis in the third case was made.\* I have never heard a diastolic murmur which I could confidently refer to the tricuspid orifice.

The *area* of tricuspid murmurs is of an irregularly triangular figure, having its base at the level of the ensiform cartilage, and extending from the middle line of the sternum to a point in the left fifth intercostal space about an inch to the inside of the apex. The vertex of this triangle would correspond to the lower edge of the left fourth costal cartilage near the sternum; and its sides to two oblique lines connecting this with the extremities of the base. Thus, the lower third of the left margin of the sternum, and the inner portion, to the extent of about an inch and a-half, of the fifth and sixth costal cartilages, and fifth intercostal space of the left side, together with a somewhat less extent of the fourth intercostal space, would be included in this area.† It therefore overlies the inner half of the mitral area; but, whereas murmurs of the latter origin have their point of maximum intensity *at* the apex, tricuspid murmurs are loudest, and perceived to be closest to the ear, from one inch to two inches internally to that point.

The foregoing description and mapping of the area of tricuspid murmurs are based upon a careful study of the three cases already mentioned. I believe they will be found in practice to define pretty accurately the limits within which tricuspid murmurs are confined, although differing in many particulars from the descriptions of previous writers. The space thus included would be a nearly equilateral triangle, and the point of greatest

\* In cases of the twofold lesion, the loudness, and usually also the quality of the murmur, will contrast strikingly at two points equidistant from, and to the right and left of, that of apex pulsation.

† See Plate Fig. II.

intensity of tricuspid murmurs would be, *quam proxime*, the centre of this space.

Of murmur arising at the orifice of the pulmonary artery I am acquainted practically with only one form, namely, that of systolic rhythm; of this I have met with eight examples, six in females, and three in males. In the former it was associated with anæmia and leucorrhæa without a single exception, and in the greater number with menorrhagia likewise (cases of Anne McK., R. E., Anne C., Anne S., Mary McC., and Miss C.). Most of these females were under twenty years of age, and one only was over forty. Two of the male patients were nervous and under twenty years; one was addicted to masturbation; and the third was the subject of carcinoma of the mediastinum engaging the root of the pulmonary artery.

As to the quality of the murmur, it was harsh in two instances, and in the remaining cases it was soft.

The point of maximum intensity, the area of diffusion, and the transmission of this murmur are of great value diagnostically.

The area of pulmonic murmur corresponds to a circle of about two inches in diameter, covering the inner portion of the second intercostal space of the left side, and adjacent portions of the second and third costal cartilages, and also, to a slight extent, the edge of the sternum.

Beyond the limits of this circle murmurs of pulmonic origin are rarely audible. Those which have occurred in my experience were in every instance limited to it; they were not traceable upwards towards the clavicle and into the neck as are those of aortic origin, and were not audible in the left back as are mitral systolic murmurs of much force. The rhythm of this murmur might readily lead the observer to mistake it for systolic murmur in the aorta; but its *abrupt* termination in the direction upwards at the level of the middle of the second costal cartilage, its not being audible to the right of the mesial line of the sternum, and its non-transmission into the carotids, sufficiently distinguish it from the latter, irrespectively of the difference as regards the point of maximum intensity in the two cases. From mitral murmurs the last-mentioned difference will serve as the chief distinction, but in a less degree



the circumstance that mitral murmurs when loud may be heard likewise in the left back will serve to distinguish it.

In none of the cases which have come under my notice, with one exception, have symptoms of organic lesion existed; the patients, with this exception, were all anæmic, venous murmur existing in the greater number, and in several the cardiac murmur ceased under ferruginous treatment. Notwithstanding, therefore, that in only one of these cases (Case 66) have I had an opportunity of examining the heart by dissection, I feel warranted in concluding that the murmur in all the others was hæmic.

Hope has never met with an example of valvular lesion in the pulmonary artery,\* nor does he allude to hæmic murmur in this vessel.

Contraction of the orifice of the pulmonary artery is a usual concomitant of permanent patency of the foramen ovale; but, in such cases, the diameter of the vessel being, by a gradual process of accommodation, simply adapted to the diminished volume of blood passing through it, and no disproportion between the vessel and the blood-stream thereof existing, murmur is not produced. It is conceivable, however, that systolic murmur in the pulmonary artery may result from compression of that vessel by a tumor of any kind. Of this case, 66 was an example.

Hope has reported one case of diastolic murmur in the pulmonary artery due to dilatation of the vessel and consequent inadequacy of the valve. He states that the proportion of valvular disease on the right and left sides of the heart is as 1 to 16 or 20, and that this lesion is by much less frequent in the pulmonary artery than at the tricuspid orifice. Walshe places cardiac murmurs of organic origin in the following order of frequency, viz., mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.†

Other causes of intracardial organic murmurs may exist irrespectively of valve-lesion. Thus, murmur may be due to

\* *A Treatise on the Diseases of the Heart*, third edition, 1839, p. 385.

† *Diseases of the Heart*, third edition, p. 108.

patency of the foramen ovale, to aneurism of one of the chambers of the heart, or to aneurism of the aorta or pulmonary artery communicating with one of these chambers. I have met with three examples of unclosed foramen ovale, in each of which I have had *post mortem* evidence of this organic defect. Two were past the middle period of life, and one was a child. In one of the former (Case 72) there was apparent hyperoxidation of the blood, the cheeks and lips being florid, and no tendency to coma. Fatty degeneration of the heart and considerable hypertrophy of the left ventricle were found to exist, and likewise patency of the foramen ovale, but no valvular disease. In this case no cardiac murmur of any kind had existed.

In the remaining two cases there was cyanosis, and in one of these (Case 71) systolic apex-murmur. In this latter case no valve-lesion existed, but the muscular tissue of the heart was in an advanced state of fatty degeneration; and to the weakness and yielding of the walls of the left ventricle during systole, arising from this cause, I am disposed to attribute the murmur of mitral reflux.

In the third case (No. 73), no murmur whatever existed. I cannot, therefore, speak from personal experience of murmur at the foramen ovale. Nevertheless, I can admit it theoretically, and, as I have elsewhere\* explained, its rhythm in my view would be presystolic.

Of aneurism of the several chambers of the heart, and of the aorta communicating with one or other of these, Dr. Thurnam has given numerous illustrative examples.† In regard to rhythm there is a dearth of reliable evidence; but, guided by my personal experience of two fully observed cases (Nos. 69 and 70), I believe it may be stated, generally, that in the former series the murmur coincides with the diastole of the chamber whence the aneurism springs, and in the latter that it is both systolic and diastolic.

I cannot agree with Dr. Walshe‡ in the opinion "that excess of force of propulsion of naturally constituted blood would

\* "Lectures on Diseases of the Heart," *Medical Press and Circular*, July, 1866.

† "Memoirs on Aneurism of the Heart," 1838; and "Spontaneous Varicose Aneurism of the Aorta," *Med. Chir. Transact.*, 1840, vol. xxiii.

‡ *Diseases of the Heart*, third edition, 1862, p. 283.

seem capable of generating direct murmur;" nor have I met with a case of hypertrophy in which mitral regurgitant murmur occurred during palpitation by disturbance of the action of the papillary muscles, as Dr. Walshe surmises. Such murmurs, he adds, are fugacious. I have met with murmur answering to this description, only in cases of weakened heart; as in those addicted to excessive tobacco smoking, or to masturbation.

Finally, I cannot recall a single authenticated example, in my own experience, of murmur at the aortic orifice arising from mere derangement of proportion between that orifice and the left ventricle, by dilatation with hypertrophy of the latter, as alleged by Walshe.\* I have invariably found at the orifice, in such cases, some structural alteration sufficient to account for the murmur.

Doctor Purser thinks that where the left ventricle is greatly dilated, as in permanent patency of the aortic valve, a murmur of mitral regurgitation may arise from valvular incompetency produced by *relative* shortening of the chordæ tendineæ; that is, by the absence of lengthening of these chords proportionate to the expansion of the cavity of the ventricle.† Such an occurrence is conceivable, but the condition mentioned would be readily detected on *post mortem* examination of the heart, more especially as the absence of lesion of the valves would necessarily lead to a careful scrutiny as to the cause of the murmur. I have occasionally met with examples of mitral inadequacy, as indicated by murmur, where neither valvular lesion nor dilatation of the orifice existed. This I have endeavoured to explain in another way (*vide antea*, p. 219), but I have never witnessed the pathological combination mentioned by Dr. Purser.

Doctor W. T. Gairdner has made some original and valuable observations on organic murmurs and their combinations.‡ He admits postsystolic murmur ("ventricular systolic") but does not make a distinction between it and murmur of systolic rhythm. He likewise virtually admits postdiastolic murmur, which, if valvular, is always due to blood entering one of the

\* *Opus citat.*, p. 284.

† *Irish Hospital Gazette*, March 15th, 1873.

‡ *Edinburgh Medical Journal*, vol. vii., November, 1861.

ventricles "either from the auricle or the artery;" in the latter case indicating reflux. In this, as in the former instance, however, he fails to distinguish between murmurs synchronous with, and those succeeding the sound. He denies entirely prediastolic murmurs.

I have met with only three examples of left auriculo-ventricular postdiastolic murmur (cases M. Coates, T. Doyle, and M. Ferguson); and this, as already stated, I believe to be due to extreme mitral narrowing.

As to the combination of mitral murmurs, I hold that of presystolic and postsystolic ("auricular-systolic and ventricular-systolic"), or that of one "running up to, with one succeeding or running off from the first sound," which implies that the first sound is perfect as quite exceptional; at least I have never met with an example. The usual combination of left apex-murmurs is that of presystolic with *substitutive* systolic murmur. Indeed it would be difficult to conceive a perfect first sound occurring where both obstruction and regurgitation exist, and therefore, by inference, disorganization of the valves.

This objection will not apply to the combination of systolic and postdiastolic murmur ("v. systolic and v. diastolic"), because the former may exist at the apex with an imperfect first sound, and the latter at the base with a more or less normal second sound; or both at the base with sounds more or less normal, the first an accompanying systolic right basic or aortic murmur, and the second a postdiastolic murmur of identical site and origin.

The combinations of organic murmurs most often met with, set down in the order of frequency in which they occur, I believe to be the following:—

1. Right basic-systolic and diastolic (aortic obstruction and reflux).
2. Right basic-systolic and postdiastolic (aortic obstruction and incipient reflux).
3. Left apex-systolic, and right basic-systolic (mitral reflux and aortic obstruction).
4. Left apex-presystolic, and left apex-systolic (mitral obstruction and reflux).
5. Left apex-systolic, and right apex-systolic (mitral reflux and tricuspid reflux).



6. Left apex-presystolic, and right apex-presystolic (mitra and tricuspid obstruction).
7. Left apex-postsystolic, and right basic-postdiastolic (incipient mitral and aortic reflux).
8. Left basic-systolic, and right basic-systolic (pulmonic and aortic obstruction).

Non-organic murmurs are divisible into two classes, viz.: *hæmic* and *dynamic*. *Hæmic* murmurs, as the name implies, are of blood origin, or due either directly or indirectly to an alteration in the quantity or in the corpuscular constitution of the blood. They may be located in the heart, the arteries, or the veins.

These murmurs are sometimes designated as *anæmic* or *spanæmic*, according to the view taken of their pathogeny; the former term implying that the murmur arises from absolute decrease in the mass of the blood, and the latter, from simple attenuation or relative reduction in its corpuscular element.

That intra-vascular murmur may be caused by decrease in the density, irrespectively of the quantity of the blood, seems proven by the observation of Richardson, that when water in large quantity is injected into the veins of dogs, a bellows murmur is developed throughout the entire vascular system.\*

A similar phenomenon may be witnessed in the human body in cases of extreme spanæmia from chronic disease of the primary organ of nutrition; and likewise in chlorosis. In these cases the volume of the blood would seem to have undergone no diminution, as testified by the character of the pulse and the fulness of the superficial vessels. Yet I have repeatedly heard in such cases a loud bruit de soufflet in the heart and principal vessels of the body, both arteries and veins.

That absolute loss of blood is competent of itself to produce murmur, is shown by the occurrence of murmur in the heart and large arteries after copious hæmorrhage.

Hope† narrates a series of experiments performed upon dogs which likewise go to establish the truth of this doctrine, and further show in a striking manner what, as being already

\* *Medical Times and Gazette*, Lecture, October, 1868.

† *A Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1839, p. 99.

well known and universally admitted, it was scarcely necessary to affirm by direct experiment; namely, that increased force and rapidity of the action of the heart intensify the murmur of anæmia, whilst weakening and slowing of it have the opposite effect.

Hope\* holds "that the murmurs and tremors, as well in the heart as in the arteries, are occasioned by modifications in the motion of the fluid, occasioning increased friction and vibration;" and, to establish this doctrine, he undertakes to prove: 1. "That liquids permeating tubes, do occasion murmurs and tremors." 2. "That in the living subject, modifications in the motion of the blood calculated to elicit murmurs and tremors, do take place under the circumstances in which such murmurs and tremors actually occur." 3. "That the explanation applies equally whatever be the circumstances under which the murmurs and tremors occur." He sustains these propositions by arguments of much force, and by experiments, and concludes that "the physical circumstances usually attending inorganic murmurs in the heart and arteries are, attenuation of the blood; unfilled arteries permitting unusual vibration of their walls and a rippling current; and a certain velocity of the current occasioned by abrupt contractions of the heart."

Andral, whilst accepting unreservedly the doctrine of remote causation of hæmic murmurs by reduced volume or diminution in the corpuscular constituent of the blood, regards as the immediate cause, and dependent upon the preceding, an alleged spasmodic constriction of the orifices at which the murmur occurs.† When, however, it is borne in mind that the orifices in question are those of the aorta and pulmonary artery, the immediate boundaries of which are fibrous not muscular in structure, few pathologists will, I apprehend, subscribe this opinion of the illustrious Andral.

Hope,‡ Beau,§ and Bellingham,|| localize the murmur at the orifice of the aorta. This is likewise the opinion of Flint.¶

\* *Opus citat.*, p. 98.

† *Notes au Traité de l'Auscultation de Laennec*, tom. iii., p. 103, Paris, 1837.

‡ *Opus citat.*

§ *Traité d'Auscultation*, Paris, 1856.

|| *Diseases of the Heart*, Dublin, 1853.

¶ *Diseases of the Heart*, Philadelphia, 1859.

Hughes\* suggests the orifice of the pulmonary artery as the seat of certain hæmic murmurs, and in the justice of this view I entirely concur. The authors just mentioned agree in regarding blood friction, intensified by attenuation or absolute loss, and accelerated movement of the blood, as the cause of hæmic murmur; and the aortic orifice as its seat when occurring within the heart.

In an article of great literary merit,† Dr. Parrot maintains that the seat of cardiac hæmic murmur is the tricuspid orifice, and its cause regurgitation of blood during ventricular systole. I am by no means convinced by the reasoning of M. Parrot; it in many particulars seems inconclusive, and the premises on which it proceeds are erroneous and contrary to clinical experience. For example, I cannot admit that jugular bruit is an invariable accompaniment of cardiac murmur of hæmic origin; nor can I endorse the assertion that such murmur is usually loudest at the inner portion of the left fourth intercostal space, and thence especially traceable upwards and to the right to a certain distance; both of which circumstances would be necessary corollaries of his doctrine as to the cause and site of such murmurs.

Walshe‡ says, "An intracardiac hæmic murmur of this variety (depending on change in composition of blood) is of moderate or very slight intensity, commonly of medium or low pitch, short or moderately prolonged, of whiffing quality, very easily rendered temporarily harsh by excitement of the heart, and modified in intensity by certain changes of posture.

"This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta, and of the pulmonary artery, with a force at each proportional to the power of its communicating ventricle; scarcely conducted along the aorta at all; frequently audible, on the contrary, at the second left or pulmonary cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex."

\* *Guy's Hospital Reports*, second series, vol. vii., 1854.

† *Archives Générales de Médecine*, Aout, 1866.

‡ *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 92.

This is a terse and accurate description of cardiac blood-murmurs as usually met with, but conveys no intimation of the author's opinion as to the cause of these murmurs.

Skoda throws doubt on the very existence of blood-murmurs. He says,\* "the opinion that murmurs are caused by a particular condition of the blood must be looked upon as hypothetical until it has been shown in what that particular condition of the blood consists. It is not true that a watery state of the blood is a cause of murmurs in the heart; I have many times abstracted very watery blood from patients in whom no murmurs existed.

"It is true that murmurs do occasionally arise in the heart after great loss of blood, and in anæmia, but not so constantly as to justify us in considering the anæmic condition of the blood as the only cause of the murmurs."

The experiments of Richardson, and the general experience of physicians, go to show, as already stated, that spanæmia is of itself quite competent to produce intracardial murmur, and even the high authority of Skoda will be held insufficient to outweigh these considerations.

Again, no well informed physician would venture to maintain that the anæmic condition is the *only* cause of anæmic murmur. It is, no doubt, in the absence of structural disease or debility, the primary and principal cause of such murmur; but, a set of conditions immediately dependent upon it intervenes, and acts as the compound proximate cause of the murmur. To these conditions attention will be presently directed.

At least two factors enter into the causation of hæmic murmurs; viz., 1. *Friction* of the blood corpuscles, (a) against one another, and (b) against the edges of the opening and the walls of the vessel; and 2. *Vibration* of the heart and walls of the vessels.

Corpuscular friction depends upon ventricular systole, and is directly as the force of latter.

\* *A Treatise on Auscultation and Percussion*; translated by Markham, 1853, p. 211.



Chauveau has shown, experimentally,\* that the rate of circulation in the carotids of a horse was as follows, viz. :

During ventricular systole, 20·4 ; in period immediately succeeding systole and coincident with closure of semilunar valves, 8·6 ; average rate of period succeeding dirotic impulse and second sound, 5·9 inches per second.

Thus there are three currents of different rates during the period of a single complete action of the heart. After the dirotic or diastolic impulse the rate grows gradually slower, till just before the repetition of the impulse (*i.e.*, during the momentum of auricular systole), when it is almost *nil*.

He also found that as the small arteries are approached the systolic impulse rapidly diminishes, and the dirotic (diastolic) impulse becomes very feeble or is entirely abolished. The *constant* flow, however, is very much increased in rapidity. Thus it would seem that by the elasticity of the large arteries the impulse, both systolic and dirotic, is gradually converted into a continuous force, under which the flow in the smaller vessels becomes constant, and by an equal distribution of that force, also more rapid ; that the current in the large arteries is intermittent ; in those of medium size remittent ; and in the very small arteries nearly constant.†

Increase of rate no less than of force of current has the effect of intensifying friction ; but, as both force and rate of circulation are at their maximum during ventricular systole, it is quite intelligible that a murmur whether organic or non-organic, depending upon blood-friction, should be systolic in rhythm ; and furthermore as, other circumstances being equal, friction must be greatest where the passage is narrowest, the seat of murmur of either kind will be that of the most constricted portion of the vessel. Now, even in the healthy state, the narrowest portion of the aorta and pulmonary artery is the orifice of both vessels ; hence the occurrence of hæmic murmur by preference in these situations.

It is worthy of remark, as affording further evidence in support of this view, that whilst hæmic systolic aortic murmur may or

\* *Nouvelles Recherches Experimentales sur les Mouvements et les Bruits Normaux du Cœur envisagés au point de vue de la Physiologie Médicale*, Paris, 1856.

† *Physiology of Man*, by A. Flint, vol. i., 1866.

may not, according to the force and efficacy of its several contributory causes, be accompanied by murmur in the arteries, the latter *never* occurs unaccompanied by murmur at the orifice of the aorta, except where caused by extrinsic pressure upon the vessel, morbid narrowing of its calibre, or roughness of its interior.

Vibration of the heart or vessels is due in part to friction ; but likewise, in some degree, to the existence of an eddy or ripple in the passing current. In organic disease this is, in many instances, sufficiently pronounced to be perceptible to the coarser sense of touch ; but in cases of non-organic murmur due to modified constitution of the blood, vibration is never detectable by the hand as fremitus, though manifest to the finer sense of hearing.

Chauveau has shown, experimentally, that when a liquid current is hurried past a constriction in its passage into a wider space beyond, a ripple or perturbation in the current is created, giving rise to vibration and murmur.

Corrigan, in 1829, clearly established this by experiments conducted upon the intestine of an animal. He says :\* “ While the intestine was tense no sound, or a murmur exceedingly indistinct, was heard ; but any part being constricted so as to produce an alteration in the motion of the fluid, a very loud bruit de soufflet immediately became evident.” I have already criticised this passage (p. 172) on the ground of its containing a denial of friction-murmur from water-currents passing under strong pressure through tubes, whether solid or not, and independently of constriction. I may add that I cannot accept the conclusion of the author that, in experiments of the kind described by him, murmur is audible only on the *distal* side of the constriction ; because the influence of friction as a factor, would be thereby in effect excluded. I have carefully repeated Corrigan’s experiments, using a human intestine under a water-pressure of several pounds to the square inch, sufficient ultimately to rupture the intestine, and have satisfied myself, and my assistants, of the existence of bruit on the *proximal* side of the constriction produced by means of a strong cord. Still,

\* *Lancet*, April 11th, 1829.

Corrigan has established the main fact of the existence of ripple, and consequent vibration and murmur, beyond the seat of constriction.

Friction, whether inter-corpuscular or vaso-corpuscular, in a degree adequate to the production of murmur in blood impoverished as to its solids, or diminished as to its absolute volume, is due mainly to atony or relaxation of the vascular walls, and low arterial tension consequent upon malnutrition of the vaso-motor nerve centres.

Vibration is, I believe, traceable to the same cause. I am glad to find that in this opinion I am fortified by the authority of M. Marey, who says: "The murmurs which anæmia, chlorosis, and fever produce at the level of the orifice of the aorta, are due to lowering of arterial tension, and the greater rapidity with which ventricular systole is accomplished."\*

In this state of the arteries, and owing to the constant and rapid oscillation of their walls, the blood does not move in an equable current, but is thrown into a series of ripples or eddies by which collision and friction are multiplied. Should constriction or other obstruction to the onward flow of the current present itself, vibration, as shown by Corrigan and Chauveau, will be intensified, and the murmur proportionately louder.

There can be no doubt that, as long since urged by Stokes,† many organic murmurs are reinforced, or even in greatest part constituted, by morbid sound arising from the friction and vibration of impoverished blood in transit through rough or constricted passages; and that the alleged cure of organic disease of the heart, and the actual improvement of the patient's health which so often follows tonic, and especially chalybeate treatment persistently continued, are to be attributed to the improved condition of the blood, and the consequent cessation of murmur *quoad* anæmia.

The *diagnosis* of hæmic cardiac murmurs has reference mainly to the distinction between such murmurs and those of organic origin; and, in most instances, may be confidently made from a careful consideration of the actual physical signs and the symp-

\* *Arch. Général de Médecine*, par le Dr. Parrot, Aout, 1866.

† *Diseases of the Heart and Aorta*, 1854.

toms exhibited by the patient. In the few instances where this is not possible, a full consideration of the antecedent history of the case, and further observation of the effect of treatment, which in doubtful cases should always proceed on the assumption of the murmur being of hæmic origin, will remove all difficulty. Cases of this doubtful nature are usually of mixed character, being due to anæmia and to structural alteration in varying relative proportions; and the degree in which each of these causes operates can be determined only by treatment. If, in any given case, abatement without cessation of the murmur, and improvement in the general condition of the patient result from the persistent use of chalybeates and of regimen adapted to the amelioration of the blood, not only may the diagnosis of a mixed case be confidently made, but the *degree* in which the humoral and the structural element had respectively operated, may be likewise satisfactorily determined by a comparison of the previous with the actual signs and symptoms.

Cardiac murmurs of hæmic origin are invariable basic, they are loudest at midsternum or in the anatomical site of the orifices of the aorta and pulmonary artery; with this point as centre they have a diffusion-area of three to four inches in diameter, according to the intensity of the murmur and the conducting qualities of the chest wall; but they never exhibit a definite line of propagation, as is the case with organic murmurs in this situation.

Not unfrequently, but only in aggravated examples, murmur likewise exists at some point of the ascending and transverse aorta, and in the carotid and subclavian arteries, but never, as far as I have observed, in unbroken continuity throughout these vessels; a feature in which they differ from organic murmurs at the orifice of the aorta. The latter are, moreover, not infrequently audible at the apex, though with diminished intensity; the former never.

Finally, in the recumbent posture feeble hæmic murmurs are in many instances inaudible, but are promptly developed when the patient sits up; they are usually soft and blowing in quality but subject to considerable variation in this respect, assuming occasionally a sharp or sawing character; whereas, organic aortic



murmur is never entirely suspended, save in extreme debility of the patient, and undergoes, with change in the posture of the body, an alteration in degree of loudness but not in quality of sound.

In the principal arteries of the body, down to those of the third order, murmur may be developed in certain pathological conditions.

Bouillaud maintains that in a state of health murmur is produced in these vessels by the shock and friction of the blood against the vascular walls at each stroke of the left ventricle.\*

According to him, arterial murmur varies with the strength of ventricular contraction, liquidity of the blood, and the thickness, tension, and internal smoothness of the arteries. Thus the physiological murmur passes by imperceptible gradations into that which is recognized as pathological, and named by him *bruit de soufflet ordinaire et intermittent*.

I cannot admit the existence of physiological arterial murmur, although *a priori* I should have expected it, bearing in mind the occurrence of murmur in metallic or elastic tubes through which water passes under strong pressure. As a matter of fact, however, I have failed to discover this phenomenon in the arteries of the healthy body;† and if speculation were not prohibited in a science of experiment, I would venture to suggest, whether its absence may not be due to the layer of white corpuscles by which normally the vessels are lined, and by which friction of the passing current against the vascular wall may possibly be obviated. Arterial murmur in the pathological sense may arise, according to Bouillaud, from, 1. Pressure on the artery in which it exists; and of this he offers as an example the so-called *bruit placentaire* which he attributes to pressure of the gravid uterus upon the iliac arteries; 2. Aneurism; 3. Roughening of internal surface of artery by calcareous or cartilaginous plates with or without narrowing; 4. Passage

\* *Traité Clinique des Maladies du Cœur*, tom. i., 1835.

† If the noise produced by sudden tension of the vessel, and friction of it against the end of the stethoscope during expansion be excluded, there is nothing to present even the semblance of murmur.

of blood from an artery into a vein (in this instance, however, the murmur is venous not arterial) ; 5. Great agitation of the arterial system as in anæmia and chlorosis.

In addition to intermittent arterial murmur, synchronous with ventricular systole and arterial diastole, which is the ordinary form in which this phenomenon is presented, Bouillaud admits three others, namely, *bruit de soufflet continu, ou à double courant* ; *bruit ou soufflement de diable* ; and *sifflement modulé, ou chant des artères*.

*Bruit de soufflet continu*, he describes as consisting of two murmurs, seemingly continuous, the first of which is very loud like that of a forge bellows, and succeeded immediately by another much less loud ; sometimes it resembles the cooing of a turtle dove, sometimes the whistling of the wind through a chink or key-hole. It occasionally passes by a gradual ascent into the *bruit de diable*, and may be always made to do so by regulated pressure.

*Bruit de diable*, so named from its resemblance to the whizzing noise made by the "diable," which is a French toy played by whipping, is regarded by Bouillaud as arterial in origin, and most frequently heard in the carotid and subclavian arteries ; usually on one side only and more frequently the left than the right ; but according to Laennec it is more common on the right side, and in this opinion I concur. Even when audible on both sides it is louder on one side than on the other. It is subject to capricious and irregular intermittences which are at present inexplicable. Gentle pressure with the stethoscope sometimes enfeebles it, sometimes converts it into a growl. It is rendered louder by turning the head to the side under examination, and enfeebled or suspended by lifting the larynx from the arteries. Prolonged effort of the person examined, and pressure on the vessel above the point auscultated, promptly suppress it.

The general accuracy of this graphic sketch of *bruit de diable* by Bouillaud I readily admit ; but, with the most recent writers on this subject, I regard the phenomenon as of venous origin, and as having its seat in the jugular veins. Indeed, it is now very generally named "jugular murmur," or "venous hum."

The fact, admitted by Bouillaud, that gentle pressure *above* the point examined will suffice to suppress the murmur, is *prima facie* evidence against his doctrine; and, still further, the now universally admitted fact that this phenomenon is all but confined to the right side of the neck, points directly to the superior cava and its tributaries as being its seat. Finally, its continuous though modulated character may be regarded as confirmatory of this view.

The musical arterial modulation described by Laennec, as consisting of "musical tones as if the artery were become a vibrating string from which two or three notes were drawn out in succession by advancing and drawing back the finger upon it,"\* distinguished by Bouillaud as *sifflement modulé ou chant des artères*, and compared by him to the buzzing of a fly, is peculiar to delicate, nervous, and anæmic subjects, as correctly stated by Bouillaud. In such subjects, usually females, I have occasionally heard it in the arch of the aorta and its primary branches, but not elsewhere; it consists of a whistling, squeaking note, variable in pitch and quality, and generally inaudible when the body is recumbent and the nervous system in a state of functional quiescence, but promptly brought out by mental excitement or sudden assumption of the erect posture. In one notable case the arteries were remarkably tortuous, and the patient was liable to sudden and unaccountable flushings of the face. This musical note ascends with the ventricular systole, and usually, but not invariably, descends with the recoil of the arteries; its precise mode of production is not known, but as to cause, it is unquestionably a nervous phenomenon.

In the remarkable case above alluded to, it was noticeable that the patient's face became suddenly flushed on my appearing at her bed-side during the morning visit, and coincidentally the murmur was unusually loud; and on visiting her again before leaving the ward I have repeatedly found the face pale, the pulse tranquil, and the murmur all but, or even quite, inaudible. Whether arterial lesion, other than the tortuous condition already alluded to, existed in this case, I cannot say; and it is probable that, as palpitation and marked acceleration of pulse always

\* Forbes' translation, p. 557.

preceded increase of the murmur, the nervous influence accessory to it operated not directly upon the vessels, but through the heart. It is not difficult to conceive that the heart propelling with increase force and rapidity successive volumes of blood into the primary arterial trunks, would, if these were flexuous, be competent to develope in them a murmur of very high pitch, synchronous with the contraction of the ventricles, and occasionally also continued into the systole of the artery.

In several instances I have heard the simple arterial souffle in the arteries of the neck and shoulder, when no murmur whatever existed in the heart or aorta. In the carotids I have heard such a murmur in the person of a nervous female, with a weak and dilated heart, a month after confinement; it existed independently of pressure, which, however, rendered it proportionately louder.

I confess I am unable to account for strictly localized and transient carotid murmur in this and many similar cases, in which no palpitation of the heart, and presumably no arterial disease existed; and certainly no enlargement of the thyroid, or other cervical tumor. Is it to be regarded as the result of local arterial inhibition through the agency of the great splanchnic, or vaso-inhibitory system of nerves?

With the subclavian arteries, however, the case is different. In these vessels I have in numerous instances heard bellows murmur in two different connexions, and of widely different import; namely (*a*) in cases of disproportionate development of the subclavian muscle, as in artificers, such as blacksmiths, sawyers, etc., who use their arms much in the pursuit of their calling; and (*b*) in the subjects of primary tubercular deposition in the extreme apex of the corresponding lung.

Doctor Richardson avers\* that he has never examined the chest of a carpenter, cabinetmaker, or sawyer, without finding subclavian murmur. He states, and very correctly, that where it exists it is always intensified by the subject under examination holding the corresponding arm horizontally or at right angles with the side, and that in many cases it is audible only under these latter circumstances. This he accounts for by

\* *Medical Times and Gazette*, 1870.



assuming compression of the artery between the subclavius muscle and the first rib.

I have repeatedly met with subclavian murmur in persons of the calling mentioned by Dr. Richardson; but in many, likewise, I have failed to find it. I have, moreover, repeatedly heard murmur in this vessel in persons whose subclavian muscles could not have been assumed to have undergone excessive development by reason of their avocations, and yet in whose lungs there was no evidence of tubercular or other solidification.

In all these cases, however, the murmur was either brought out or intensified when the patient held out his arm, horizontally, for a few moments, by the action of the muscles of his shoulder. I am therefore of opinion, that Dr. Richardson's theory is correct *quoad* his explanation of the abstract phenomenon. I believe that murmur is neither audible in *all* who pursue the handicrafts which he has named, and that it is not limited to such exclusively; although undoubtedly of more frequent occurrence in the trades which he has mentioned than in any others. I am fully convinced of murmur due to this cause. In regard to the absolute proportion of *all* cases in which subclavian murmur occurs, and the influence of sex and age in its production, Dr. Richardson states\* that he has met with it in 51 cases out of 2,000: *i.e.*, in 2.55 per cent. Of these 2,000 patients, 1,011 were females, and 989 males; yet the murmur existed in 48 of the males, and in only 3 of the females; the youngest patient presenting this murmur was aged 18, and the oldest 74 years. It would thence appear that subclavian murmur is of much more frequent occurrence in the male than in the female sex, and that it may be present at any period of life from puberty to old age.†

Subclavian murmur in persons of phthisical proclivity is of much greater diagnostic and practical importance. In these it exists usually on one side, and only at the acme of inspiration. During this portion of the respiratory act it is remarkably loud, and is repeated once, twice, or oftener, according to (*a*) the dura-

\* *Loco citat.*

† Dr. Leared (*Medical Times and Gazette*, October 31st, 1868,) prefers to attribute arterial murmur, generally, to local arterial contraction from reflux irritation.

tion of the inspiratory effort, and (b) the rate of pulsation of the heart, corresponding exclusively with ventricular systole. Subclavian murmur having the connexion and characters now mentioned, viz., co-existence with certain presumptive symptoms, with or without other physical signs of incipient pulmonary phthisis, and being audible at one side, at the acme of inspiration only, irrespectively of the position of the corresponding arm, I regard as strong evidence of solidification of the apex of the lung. I am limited for confirmation of this view, however, to observation of the subsequent progress of only a few such cases, and have not had the advantage of a *post mortem* examination of the body in any. Nor is it difficult to afford a rational explanation of the phenomenon. At the acme of inspiration the apex of the lung glides forwards beneath the arch formed by the subclavian artery in its first and second stages, brushing its anterior surface forcibly against that vessel in its ascent.

If in this situation, namely, where the imprint of the vessel in the shape of a semicircular indentation on the anterior surface of the apex may be observed in the healthy lung, the pulmonary structure has become solid, it is conceivable that a murmur in the vessel may result from pressure made upon it by the solid portion of the lung, at the moment when the parts are brought into mutual contact, by ascent of the apex in inspiration.

*Venous murmur* is a phenomenon of considerable speculative interest; it has afforded, and still continues to afford, subject matter for the most ingenious and diverse speculations. It was first observed by Laennec, who compared it, not inaptly, to the roaring of the sea heard at a distance; and, still more appropriately, to the continuous but undulating sound heard on applying a spiral sea-shell to the ear. Laennec, however, located this phenomenon in the carotid arteries, alleging that it was occasionally audible likewise in the heart. This opinion was adopted and advocated, with much force and ingenuity, by Andral, Bouillaud, Corrigan, Laharpe, Vernois, Marey, and Beau.

In 1837, Dr. Ogier Ward,\* of Birmingham, propounded the doctrine of the venous origin of the continuous cervical murmur,

\* *Medical Gazette*, vol. xx., p. 7.

up to that time regarded as having its seat in the arteries of the neck.

Hope adopted this opinion, which was subsequently professed by Hughes, Bellingham, Walshe, and Stokes. In France, Aran, Moneret, Hardy and Béhier, Chauveau, Barth and Roger, and Parrot, advocate the same view, which is now all but universally held by physicians.

Laennec regarded this murmur as a purely nervous phenomenon, and due immediately to spasmodic contraction of the arteries.

Bouillaud considers that whilst several causes contribute to its production, the most potent is friction of the blood against the walls of the vessel when that fluid has undergone changes of elementary constitution; and he surmises that the proximity of the heart, larynx, and trachea may have some share in its production. He admits, however, his inability to assign to each of these alleged causes its due value, or to declare that other and still unknown causes may not co-operate to produce this concrete phenomenon.

Andral, with much reserve, held the opinion that the murmur was due to the friction of an impoverished blood, projected with inadequate force by a weakened left ventricle into arteries partially contracted.

Beau holds to an alleged disproportion between the volume of the blood and the containing arteries, and consequent friction.

Bellingham denied the existence of venous murmur, save when pressure is made with the stethoscope upon the vessel, and its walls are thereby made tense.

Walshe, likewise, considers pressure upon the vein indispensable to the production of murmur within it; although, with Hamerngk and Skoda, he holds that two predisposing conditions must exist, namely, a state of permanent dilatation of the vein, and a reduced volume of blood circulating through it.\* The former of these conditions is presented by the internal jugular, only at the point where it debouches into the innominate vein. Here the walls of the vessel are kept permanently on the stretch,

\* In the fourth edition of his book (1873), Dr. Walshe does not reaffirm this theory. Indeed, with the single exception of spanæmia, he declines to associate the phenomenon with any definite condition of the veins or their contents.

and prevented from collapsing, by the deep cervical fascia, which enfolds the vein, and is attached to the fixed points of the clavicle and sternum below, and to the omohyoid, the sternohyoid, and sternothyroid muscles on either side.

The reduced stream of blood descending from the vessel above, into this dilated and patulous portion of it, assumes a spiral course; and, by the exaggerated friction and vibration of the tense walls of the vessel thereby produced, becomes a cause of murmur and fremitus.

This is an exceedingly ingenious theory, but at least two fatal objections may be urged against it; namely, the *necessary* assumption of a reduced volume of blood, and the *necessary* consequence of frémissement. It is notorious that venous murmur may be associated with a simply spanæmic state of the blood, irrespectively of decrease of its mass; and no less so, that in a large proportion of the cases in which it is heard no fremitus can be detected.

Marey,\* who adheres to the doctrine of the arterial origin of the murmur in question, regards anæmia as a remote or indirect cause of the murmur by lowering arterial tension; in consequence of which the capillaries permit a more ready outflow. Hence, when pressure is made with the stethoscope on one of the large arteries, the portion of the vessel beyond the point subjected to pressure is rapidly emptied of blood through the capillaries, or tends to be; its walls become relaxed, vibrate, and produce murmur.

It is not necessary to combat this doctrine, which is based upon an erroneous view as to the seat of the murmur; otherwise it would be sufficient for this purpose to remark, that the murmur is much louder on the cardiac than on the distal side of the point of pressure.

M. Vernois† thought the murmur arose from friction of the blood against the folds which were formed in the walls of the arteries, consequent upon reduction in volume of the contained blood.

M. De La Harpe, considering only the density of the blood,

\* *Physiologie Médicale de la Circulation du Sang*, p. 476, Paris, 1863.

† *Etudes Physiologiques et Clin. des Bruits des Artères*. (Theses de Paris, 1837, No. cccclxxviii.)



compared the arterial current to that of a column of air in a pipe, yielding, like the latter, sonorous vibrations.

M. Aran,\* regarding continuous and musical murmurs as of venous origin, attributed them to increased rapidity of circulation, and to friction, both intrinsic and against the walls of the vessel, consequent upon impoverishment of the blood. Such was likewise the opinion of Barth and Roger.

M. Chauveau,† holding these murmurs to be venous, attributed them to the vibration of a central current, or "fluid vein," which is formed in the roots of the venæ innominatæ, owing to the inability of this portion of the vessels to accommodate itself to the reduced volume of the blood (see page 183).

M. Heynsius,‡ considers vascular murmurs, in general, due to a peculiar movement of blood in a dilated portion of the vessel, and beyond a constriction. He holds that local contraction in the calibre of a vessel, must create eddy and murmur in the dilated portion in advance of the constriction.

M. le Dr. Parrot§ concludes from his observations that venous murmurs in the neck, both intermittent and continuous, are due to vibration of the blood-corpuscles produced by the passage of a current, afflux or reflux, through the embouchure of the internal jugular vein.

When the murmur is intermittent, it is synchronous with auricular contraction and jugular pulsation, and is caused, according to Parrot, by the passage of a reflux current, under the pressure of auricular systole, from the temporarily distended vena cava into the relaxed jugular; the orifice of debouchment of the latter vessel being now incompletely closed by its valves.

The continuous murmur, or bruit de diable, is composed of two elements; namely, that just described, and another, less loud, which immediately succeeds it, and which is itself caused by the onward flow of blood from the jugular, now distended by the

\* *Opus citat.*

† "Études pratiques sur les Murmures Vasculaires ou Bruits de Souffle, et sur leur valeur Séméiologique." (*Gazette Médicale de Paris*, 1858, p. 247, *et suiv.*)

‡ *Journal de la Physiologie*, tom. iii., p. 722.

§ *Archives Générales de Médecine*, 1867, tom. i., p. 649, *et suiv.* To the learned author of the last quoted article I am indebted for many of the preceding references, and for much valuable information on this subject.

preceding auricular systole, into the cava, relaxed coincidently with the commencement of auricular diastole.

It would be difficult to explain why, in this view of its causation, venous murmur should not exist in every case of engorgement of the right chambers of the heart. Yet, such is notoriously not the case; whilst, on the contrary, venous murmur is usually unassociated with venous engorgement. Besides, careful attention will show that the murmur is one of afflux exclusively. Neither can I assent to the doctrine of M. Parrot, that few persons, even in good health, are free from venous murmur; in fact, that its presence is the rule, and its absence the exception.

Hope has taken a more correct view of the conditions and cause of venous murmur, regarding it as a symptom of anæmia; under which term he includes not only diminution of volume, but likewise attenuation of the blood. According to him, it is immediately due to the twofold cause, of ripple of the current, and vibration of the walls of the vessel; and few persons exhibiting any degree of anæmia are free from it. He does not, however, attempt to explain the connexion of these proximate causes of murmur with a simply hydræmic condition.

Hope very correctly states that the ordinary venous murmur is a continuous bruit de soufflet, but liable to augmentations due to the *saccade* of the adjacent artery, and to certain modifications of tone from pressure upon the internal jugular vein, in which the murmur is most frequently located. The external jugular, and, indeed, all the large and medium-sized veins of the body may, however, be the seat of murmur in aggravated anæmia.

The rhythmical augmentations of the murmur very generally met with, coinciding as they do with ventricular systole, were regarded by Bouillaud as constituting the second element of a duplex arterial murmur. By gentle pressure upon the vein, the murmur may be so altered in quality as to resemble the noise made by a child's humming-top; this is the "venous hum" of Hope; a designation certainly more intelligible, at least outside France, than the synonyme "bruit de diable." Occasionally, and to appearance capriciously, the murmur undergoes a still further

elevation of pitch, and assumes a musical character ; aptly compared by Hope to a delicate whistle produced by the lips, the noise made by the wind passing through a key-hole or crevice, the singing of a kettle, or the song of a mosquito, in different examples. In all these cases, I have no doubt that the variation of murmur is due to alteration in the rate of circulation, of vascular pressure, or of tension of the walls of the vessel, separately or conjointly ; and consequent upon an insensible movement of the body, contraction of adjacent muscles, tension of the integument, or vaso-motor influence operating on the heart or directly upon the vein.

Respiration exercises a marked influence upon jugular murmur. As stated by Hope, it is augmented by a full inspiration, when it assumes a *rushing* character ; and by a prolonged or forced expiration, it is all but, or even entirely suspended. Gentle pressure above the point auscultated renders the murmur louder ; and strong pressure, above or below this point, has the effect of altogether suppressing it.

I agree with Laennec and Hope in the opinion that venous murmur is always louder on the right than on the left side, the authority of Bouillaud to the contrary notwithstanding ; but, I cannot subscribe the statement of Hope, that in *all* cases where it is audible on the right, it is likewise, though in a less degree, present on the left side. I have repeatedly noted its absence on the left side, when distinctly audible, though not loud, on the right.

Niemeyer\* says the bruit de diable is louder on the right than on the left side, and that it ceases when the patient assumes the recumbent posture and makes a full inspiration. He adopts the hypothesis of Hamerngk and Skoda, as to the mode of its production ; viz., that it is due to a twofold cause : permanent expansion of the internal jugular at its termination in the innominate vein, and a vortigenous movement of the diminished blood-current in passing through this portion of the vessel, and consequent vibration of its tense walls. The objections already urged against this theory, are not in any degree

\* *A Text-Book of Practical Medicine*, translated by Humphreys and Hackley, New York, 1869, vol. ii., p. 723.

lessened by the sanction it receives from the high authority of Niemeyer; it is no less characteristic of simple spanæmia when the blood has suffered no diminution of volume, than it is of the condition induced by absolute loss of blood. Nor can I assent to the statement of Niemeyer, that venous murmur may be induced in persons free from any abnormality of the blood, by causing them to rotate the head towards the opposite side; when, as he believes, the pressure made upon the vein by the omohyoid muscle becomes a cause of murmur. I have never heard genuine venous murmur in healthy persons under the circumstances just mentioned, nor as a consequence of simple pressure upon the vein, however made.

Bruit de diable, then, I regard as a sign of absolute reduction of volume, or of attenuation of the blood. It is frequently absent on the left side, and, when present on both sides, is always louder on the right. It is intensified when the patient changes from the recumbent to the sitting posture, and, for a moment, ceases to be audible on resumption of the former of these postures; but, after a brief interval, it may be again heard, though with diminished intensity. It is louder during inspiration than during expiration, and by holding the breath, is entirely suspended. Light pressure upon the jugular above the clavicle, whether made with the stethoscope, the finger, or through the muscles by rotation of the head, intensifies the murmur, whilst stronger pressure entirely stops it. It gradually ceases under appropriate treatment directed to the faulty condition of the blood, and is, on the contrary, made more distinct by causes which aggravate that condition. It is remarkably variable, and apparently capricious in regard to pitch, tone, and absolute loudness; but these variations may in every instance be traced to difference in the rate or force of the heart's action, or to variation of tension in the walls of the vein itself, under vaso-motor or vaso-inhibitory influence.

Venous murmur, though most frequently heard in the jugulars, is by no means confined to these vessels; it is present in aneurismal varix, and in the placental and uterine veins in the latter months of pregnancy. I have likewise repeatedly heard it in the sinuses of large myomatous tumors of the uterus, and once,



in the dilated veins ramifying upon the surface of a large osteosarcomatous tumor of the arm of a young anæmic man, subsequently operated on, successfully, by my friend and colleague, Mr. Tyrrell. Indeed, in anæmic subjects, gentle pressure on any of the large veins will suffice to elicit this murmur.

As to the precise mode of production of these murmurs, it may be positively affirmed that they are, like arterial murmurs, due to the multiple cause of mutual collision of the blood-corpuscles, friction of the blood against the walls of the vein, and vibration of the latter. As to why these causes operate with effect in the anæmic or spanæmic condition exclusively, it were vain to attempt an explanation in the present state of knowledge; as the inadequate and inconclusive character of the conjectures, previously referred to, sufficiently testifies.

*Dynamic* murmurs are exclusively cardiac, and located at the auriculo-ventricular orifices; they are presumably attributable to irregular contraction of the ventricles, and imply the absence of permanent dilatation or valvular disease. All writers agree in regarding them as exclusively regurgitant, but two theories are held as to the precise mode of their occurrence; namely, that they are the result, upon the one hand, of disorderly or excessive contraction of the papillary muscles; or, upon the other, of asynchronous or imperfect contraction of these, or of a limited portion of the ventricular wall. The former of these theories has been applied in an especial manner to explain the evanescent murmur of chorea, whilst the latter has been adduced to account for the murmurs so frequently met with in adynamic fever, and in nervous exhaustion.

Laennec was probably the first who directed attention to murmurs of this class. He says,\* "I can state with certainty that the bellows sound of the heart is very often met with when the organ is perfectly healthy." This statement applies, no doubt, equally to anæmic and dynamic cardiac murmurs, which were not distinguished by Laennec, and were alike attributed by him to spasm of the heart.

Dr. Todd, who, so early as 1843, insisted on the connexion between chorea and rheumatism, reiterated this opinion with

\* Forbes' translation, p. 559.

much emphasis in his Lumleian Lecture in 1849.\* In this lecture he dwells upon the frequent occurrence of mitral systolic murmur in chorea, and regards it as due to organic valvular lesion, arising from "insidious endocarditis" of rheumatic origin. Thus, he considers rheumatism to be the starting point of chorea, but mitral organic lesion to be a necessary link in the chain.

It is not possible, however, to accept this as a satisfactory solution of the difficulty; because, as Dr. Todd himself remarks, the natural tendency of chorea is to get well. He adds, "it is rare to meet with any symptom of heart affection either preceding or accompanying the chorea;" and this, notwithstanding the admittedly large per-centage of choreics in which systolic apex-murmur exists.

It would seem that the causal connexion of rheumatism with chorea, was recognized at Guy's Hospital so early as 1802.† In 1837, Dr. Bright advocated a close and immediate connexion between pericarditis and chorea. In 1839, he gave examples of various forms of muscular spasm, including chorea, dependent upon pericarditis, and immediately due, as he believed, to peripheral irritation of the phrenic nerves.‡

Dr. Bright, it is true, did not advert to endocardial inflammation or murmur in connexion with chorea, although in one of his cases endocarditis actually existed; but his observations are important, collaterally, as a contribution to the theory of choreic mitral murmur.

In 1850, Dr. Botrel declared§ that chorea was only a special manifestation of rheumatism; and in the following year (1851), Dr. G. Sée went further, holding that in nearly every case of chorea rheumatic symptoms were manifested; in many, however, only in the form of pain.

Trousseau|| only contends for "a law of coincidence," as between rheumatism and chorea; holding that while either may take precedence, rheumatism generally does so, and that chorea actually complicates rheumatism in the proportion of one-third

\* *Medical Gazette*, vol. viii., 1849, p. 663.

† Bright, in *Med. Chirurg. Transactions*, vol. xxii., 1839.

‡ *Loco citat.*

§ *Chorea considered as a Rheumatic Affection*, Special Thesis, quoted by Trousseau.

|| *Lectures on Clinical Medicine*, New Sydenham Society, 1868, vol. i., p. 395.

of the cases. Under the head of rheumatism, however, he admits without reserve, endocarditis; assuming, with Bouillaud, that every case of endocarditis is of rheumatic origin. So firmly rooted is this opinion, that, forced to admit the occasional occurrence of endocarditis in the course of scarlatina, he would save his doctrine by alleging that rheumatic arthritis is a common accompaniment of scarlatina. I have repeatedly observed chorea unconnected by history or symptoms with rheumatism, scarlatina, or valvular murmur. That articular pains are not of frequent, or indeed of more than exceptional occurrence in scarlatina, and then only in the stage of desquamation and as a consequence of indirect exposure of the limbs to cold, is my firm belief.

Doctor James Begbie\* gives three family histories illustrative of the association of rheumatism, acute inflammation of the pericardium or endocardium, and chorea, either in the same person, or by hereditary transmission. These cases are not, however, conclusive in support of a necessary connexion or kindred association of these affections. In some of the examples neither rheumatism nor cardiac disease existed; and chorea was directly traceable to fright or imitation, both very common causes of the affection. Dr. Begbie is of opinion that the connexion between rheumatism and chorea is to be explained by reference to the morbid condition of the blood in the former of these diseases.

Babington thinks that chorea, following affections of the heart or pericardium, is due to reflex irritation through the cardiac nerves and ganglia; but he admits the possibility of inflammation of the spinal meninges.†

Doctor Burrows‡ also explains the connexion by reflex irritation of the phrenic and pneumogastric nerves.

Sir T. Watson surmises that the connexion between chorea and cardiac disease may be due to their common dependence upon the rheumatic diathesis. In virtue of its tendency to invade fibrous structures, rheumatism may give rise, at the same time, to acute inflammation of the membranes of the spinal cord and of

\* *Contributions to Practical Medicine*, 1862, p. 68.

† *Guy's Hospital Reports*, vol. vi., p. 418.

‡ *On Disorders of Cerebral Circulation*, section vii.

those of the heart; and thus become the cause of chorea by mechanical irritation of the roots of the spinal nerves through inflammatory exudation and thickening of their sheaths; and also of pericarditis or endocarditis, or of both. The gravamen of the disease may fall on the spinal cord or the heart, separately or consecutively. He suggests, also, that the cardiac lesion may be an excentric cause of the spasmodic disorder of the muscles.\*

In the last edition of his work, the distinguished and veteran author modifies his former opinion; abandoning it, in effect, in favour of Kirkes' doctrine, in support of which he dwells upon the preponderance of *mitral* over other cardiac murmurs, as proving the existence of organic lesion of the valves. He adds: "I, for one, accept the conclusion that has been drawn from them (the facts and reasonings which connect chorea with valvular disease on the left side of the heart), namely, that the chorea is often, not always, the ultimate result of the injection of molecular particles of fibrine into some of the minute arteries or capillaries of the nervous tissue."†

Doctor Kirkes denies that the mitral reflux murmur of chorea may be due to choreic or irregular action of the papillary muscles, on the ground that "there is no good proof that involuntary muscular organs participate in the choreic disorder." There is much force in this objection; but, the admitted co-existence of rhythmical action of the substance of the heart, with this alleged tetanic and irregular contraction of the papillary muscles, which, as proved by dissection, are directly continuous with the fibres of the ventricular walls, constitutes a still stronger objection. We should be forced to admit, on the assumption of this theory, that whilst one and the greater portion of the length of certain muscular fibres contract with perfect order and regularity, the remaining and smaller portion of the same fibres act spasmodically, and out of harmony with the former. But no physiologist, bearing in mind the unity of nerve-centres, and the community of nerve-distribution enjoyed by both portions of the same fibres, could admit such a doctrine.

\* *Lectures*, vol. i., p. 664.

† *The Principles and Practice of Physic*, fourth edition, 1871, vol. i., p. 674.



Doctor Kirkes urges the opinion,\* and defends it with much ability, that chorea is directly due to organic disease of the mitral valve. He believes that rheumatism has a causal relationship to chorea, only so far as it contributes to the production of endocarditis on the left side of the heart; and, in confirmation of this view, dwells upon the facts that chorea shows itself only towards the close of the rheumatic attack, or even weeks or months after it; that there has been no evidence of a rheumatic tendency in at least one half of the fatal cases of chorea which he has witnessed; and that valvular disease of the heart may arise, irrespectively of rheumatism, from pyæmia, the exanthemata, pregnancy, and other causes. In chorea, moreover, endocardial inflammation engaging the valves of the left side, and giving rise to granular vegetations upon them, is a very frequent, if not an invariable, accompaniment of the nervous affection. He admits, however, that in rare instances chorea may occur independently of endocarditis; but he holds that in all rheumatic cases endocarditis is a necessary link, the cause being twofold; viz., contamination of the blood by the rheumatic or inflammatory products, and obstruction of the capillaries by disintegrated fibrinous deposit.

One statement of Dr. Kirkes I deem it necessary to give in his own words, for the purpose of recording my dissent from it. He says: "One important point in connexion with these choreic murmurs requires to be especially noted, namely, that absence of murmur is no proof of the absence of even serious organic disease of the valves of the heart. I have repeatedly observed cases in which the most careful examination failed to detect a murmur, even up to the last day of life, yet in which after death there were unmistakable signs of recent acute mischief about the valves;" and, by way of explanation, he adds, "The thickening, swelling, and other changes in the mitral valve, including fibrinous depositions, were, in the cases to which I allude, observed principally, if not exclusively, on the auricular surface of the valve, above the free margin, which was thus uninterfered with in its power of closing and preventing regurgitation."

\* *Medical Times and Gazette*, 1850 and 1863.

The accuracy of the observation upon which the preceding statement is made, I have no right to question. I can only express my astonishment, and declare that it is utterly at variance with all I have learned on this subject.

I have never witnessed a case in which *post mortem* examination showed the existence of "recent acute mischief," such as "thickening, swelling, and other changes in the mitral valve," much less "fibrinous depositions," upon it, in which at least faint murmur, or characteristic softening and prolongation of the first sound, had not been present at some period of the patient's last illness. Absence of this physical sign on the last day of life, or even for several days preceding death, owing, no doubt, to asthenia of the heart, is not uncommon; indeed it is rather the rule than the exception. But Dr. Kirkes' statement, however, goes much further than this.

I cannot, I repeat, accept the dogma; nay, I strongly and entirely dissent from it, "that absence of murmur is no proof of the absence of even serious organic disease of the valves of the heart."

I maintain that, whilst the *presence* of murmur affords no proof of the existence of structural alteration of the valves, the *absence*, throughout the patient's illness, of murmur or its equivalent prolonged and modified first sound, does afford negative proof in regard to it. In other words, I maintain that whilst murmur may be due to causes other than valvular disease, that the presence of the latter, in a degree capable of deranging health and appreciable to the eye of the anatomist, must, and actually does, always give rise to murmur as long as the heart acts with ordinary vigour.

Hence it will be seen, that I am likewise in disagreement with Doctor Markham, when he says:\* "Neither is the absence of murmur any certain indication of absence of valvular disease," etc.

On the whole question here discussed I may remark, that in several examples of chorea given in the sequel, no cardiac murmur, nor other evidence of cardiac disease existed; and that in still greater number, examples are recorded of disease of the mitral valve unaccompanied by chorea.

\* *Diseases of the Heart, their Diagnosis, Pathology, and Treatment*, 1856, p. 188.

Doctor Handfield Jones had only one example of valvular cardiac affection in fifteen cases of chorea, and in this exceptional case rheumatism had not preceded. He is of opinion that rheumatic affections rarely tend to choreic complication; but, he admits that chorea and rheumatism are allied, in being both associated with a paretic condition of certain portions of the nervous system; viz., chorea with paresis of the musculo-motor, and rheumatism with paresis of the vaso-motor nerves.\*

Doctor Hughes, after discussing with great ability the inferences to be drawn from the details of one hundred cases of chorea published by him, in regard to the alleged dependence of that affection upon rheumatic structural lesions of the spinal cord, remarks: "It seems at least doubtful, whether, in most of such cases, there exists anything more than a sympathetic affection of the spinal marrow, as these cases are curable by the same means as others."†

Doctor Ogle expresses a similar opinion, adding a number of objections of great weight, as against the doctrine of the rheumatic origin of chorea.‡

The frequent occurrence of cardiac murmur in connexion with chorea; the no less frequent association of rheumatism or the rheumatic diathesis, with that disease; and the occasional concurrence of rheumatic arthritis and organic valvular lesions of the heart in choreic subjects, have led many eminent pathologists, as previously shown, to believe that chorea is essentially a rheumatic neurosis, dependent upon eccentric irritation of the heart by rheumatic exudation or deposit upon its valves or external surface; or upon centric irritation of the cerebro-spinal axis itself, by vascular engorgement and thickening of its envelopes, inflammatory softening of its substance, or impairment of its nutrition by capillary embolism.

Is organic lesion of the heart, valvular or pericardial, a usual concomitant; or rheumatism, latent or manifest, a necessary, or even an ordinary precursor of chorea? I confidently answer both these questions in the negative.

\* *Clinical Observations on Functional Nervous Disorders*, 1864.

† *Guy's Hospital Reports*, vol. iv., 1846.

‡ *British and Foreign Medico-Chirurgical Review*, vol. xlvi., 1868.

The great majority of my cases of chorea have been in female children under thirteen years, who either have never had rheumatism, or had it at so remote a period, followed by a long interval of general good health, that it could not be regarded as in any degree causative of the chorea, in the absence of organic cardiac disease. In the greater number there was no cardiac murmur whatever; and no evidence, of any kind, of organic disease of the heart or pericardium. In a few instances a systolic bellows murmur existed at the apex; but, unattended with dyspnoea or visceral engorgement, and quickly removed by treatment appropriate to the nervous affection. In nearly every instance, likewise, the attack was traceable to fright or other emotional excitement, or to the presence of intestinal worms. In a few examples the attack was immediately preceded by articular rheumatism, with or without engagement of the heart or pericardium.

Whilst engaged in collecting materials for this work, I did not deem it necessary to record all the cases of chorea in which no evidence of cardiac disease existed. I have, however, preserved notes of a few, which I shall now furnish in abstract. In every example which has come under my notice within the last five years, I have examined the heart, physically, with great care, with a view to determine whether cardiac disease of any kind was present.

Case 1. Mary K., aged twelve years, of sandy complexion and florid face, admitted June 21st, 1869; the daughter of a poor man who, about a year previously, had been an inmate of the hospital, suffering from disease of the aortic valve which has since proved fatal. She had rheumatism the previous Christmas, which confined her to bed, and lasted one month; began to get "fidgetty" three months subsequently. She droops to left side, and admits weakness of left arm and leg; tongue furred, and suddenly retracted after protrusion; is very fidgetty even in sleep, frequently starting up. Pulse 108, regular; heart's action and sounds normal; *no cardiac murmur*. To have liquor arsenicalis (℥ v. ter die) after meals, and cold shower baths. June 28th; is much better, less fidgetty. To continue medicine at her home. Owing to scarlatina having appeared in the ward, she was discharged.



Case 2. John M., aged seven, admitted June 13th, 1869. No history of rheumatism ; is very fidgetty and droops to right side. Choreic symptoms well marked, but not in an aggravated form ; *no cardiac murmur*. To have arsenical solution and iron. Discharged cured, June 28.

Case 3. Julia S., aged nine, anæmic, consulted me as an extern patient of the hospital, October 1st, 1869. Her health had been good up to eighteen months previous to the above date ; and she never had rheumatism, or passed worms. For the last year and a-half she has been the subject of aggravated chorea. The attacks are in some measure paroxysmal, and during the period of their continuance she cannot speak, and the lips are bitten. Bowels regular ; deglutition perfect ; pulse 120, regular ; action and sounds of heart normal, and *no cardiac murmur*. Muscular power equal on both sides of body. Another child of the same parents, seven years old, has also had chorea, of which she has been cured. To have syrup of iodide of iron and Fowler's solution. Discharged, cured, after a few weeks' treatment.

Case 4. Justin McS., aged nineteen, lithographic printer, admitted February 16th, 1871. Had rheumatism, but without cardiac complication, three months previously, for which he was treated in Baggot-street Hospital. Was noticed to be fidgetty one week after leaving that institution cured ; is now sleepless, constantly biting his lips, tossing his arms and legs, and fidgetting with his fingers. Pulse weak, irregular, 96 ; cardiac action irregular and intermittent, but *no cardiac murmur* ; sight weak. To have sulphate of quinine and strychnia. Discharged, scarcely improved, after a few weeks' residence in hospital. I subsequently saw this patient in Steevens' Hospital in the same condition as above described.

In rheumatic endocarditis the mitral is the valve engaged in most instances. Yet, in the list of one hundred cases of chorea published by Dr. H. M. Hughes, there were only nine examples of permanent mitral murmur, and, of these, three only were of rheumatic origin. There were, in addition, two examples of temporary mitral murmur, and two of basic murmur, whether temporary or permanent is not stated. Out of fifty-five cases in

which a cause was assigned for the chorea, fright was the imputed cause in thirty-four instances.

Simple rheumatism was the alleged cause in only five cases; rheumatism with scarlatina in one case, and rheumatism with pericarditis in one.

In Dr. Ogle's Report,\* a list of ninety-six cases is furnished, out of which cardiac murmur presumably existed in fourteen instances.† Rheumatism is mentioned as the cause of the chorea in nineteen instances; intestinal worms in fourteen; fright in twenty-three, and menstrual derangement, or sudden arrest of the menses, in eight cases.

Out of a total of one hundred and nineteen cases of chorea published by Hughes and Ogle, in which the origin of the disease has been traced to an assignable cause, fright was the imputed cause in fifty-seven instances, or nearly one-half.

In these cases it cannot be reasonably supposed, in the absence of a rheumatic history, past or present, that rheumatism had any share in the production of the chorea. Rheumatism is mentioned as the cause in only twenty-seven out of the total of one hundred and nineteen cases; and valvular lesion of the heart can have existed in only twenty-three of the whole number, even if we include those cases in which its existence was doubtful.

From the evidence thus summarized, and from my personal observation of chorea, I feel warranted in concluding that it is not a disease of rheumatic origin; neither is it due to valvular lesion of the heart, rheumatic or non-rheumatic. I regard chorea as consisting, essentially, in a state of dynamical instability of the spinal cord, manifested by hyper-impressionability, or excessive irritability of that nerve centre. The sentient and motor tracts within the cranium are likewise engaged, but not the centres of thought or volition. The disease seems limited to the emotional centres, and the conducting media of sensation and voluntary motion.

Anæmia and general debility constitute important factors of

\* *Loco citat.*

† In all the examples where valvular lesion was actually found, I conclude that murmur had been detectable, though not mentioned in the returns. My calculation of totals, which is based upon Dr. Ogle's returns, differs somewhat from his.

chorea, but only as predisponents; and to the state arising from this twofold cause, is due the murmur at the apex, or the base of the heart, when not of direct rheumatic origin.

The basic murmur, when not the result of rheumatic disorganization at the aortic orifice, is purely hæmic, and limited, as such. The murmur so frequently encountered at the apex in chorea, is of strictly dynamic origin, and is caused by atony and partial yielding of the walls of the left ventricle at the acme of systole, as will be presently explained. Rheumatism, by inducing a state of general debility, anæmia, and muscular atony, but to this extent only, may be a remote cause of chorea, and of hæmic or dynamic cardiac murmur, associated or not with chorea; but, inasmuch as the latter association, by dependence on a common cause, is of very frequent occurrence, the mistake is not unfrequently made of assuming the murmur to be organic, and the assumed lesion to which it owes its origin to be the cause of the chorea.

Doctor Walshe\* says: "The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class. Thus, a systolic murmur, free from harshness, and of medium pitch, having all the attributes of that denoting mitral regurgitation, is occasionally heard in cases of chorea at the left apex. This murmur disappears as the primary neurotic disease itself disappears; its intensity varies with different beats of the heart; its quality similarly changes; and it may even temporarily become inaudible. The murmur in question is not localized as a hæmic cardiac murmur would be; it cannot be referred to organic change in the mitral valve, seeing that it eventually totally disappears. Nor in many of the cases which I have seen, was there the smallest ground for referring it to latent Bright's disease, or to rheumatic irritation of the endocardium.

"The most plausible hypothesis seems to be that irregular and occasional reflux takes place at the mitral orifice, through disordered action of the muscular apparatus connected with the valve. Just as the external choreal movements sometimes

\* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 95.

cease for moments or for minutes at a time, so the cardiac murmurs disappear.

"The main objection to this hypothesis seems to arise from the difficulty of understanding how the heart as a whole can act regularly (as it certainly does in chorea), and yet part of its substance contract irregularly. Still the same contradictory state of things may sometimes be witnessed in the voluntary muscles, portions of which may twitch automatically whilst the remainder of their substance is quiescent.

"The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs attending dilated hypertrophy of the left ventricle, and disappearing under treatment. So, too, may be explained a fact, for which it seems otherwise impossible to suggest a plausible theory, that in certain cases of soft, flabby, and possibly fattily atrophous heart, a murmur at the left apex will occasionally be heard, though as a rule completely absent."

Here Dr. Walshe distinctly adopts, though manifestly with misgivings as to its soundness, the doctrine of musculo-papillary spasm, in explanation not only of choreic cardiac murmur, but also of that which is occasionally heard at the left apex in flabby and fatty hearts, and of which no other theory, satisfactory to him, has been offered.

I have already urged a strong, and, as it seems to me, insuperable objection, in terms admitted by Dr. Walshe himself, to the hypothesis of spasm of the papillary muscles in chorea. This objection applies with even increased force to its admission in explanation of dynamic murmur in soft and weakened hearts, in connexion with which no evidence of nervous excitability exists; and, with great respect for Dr. Walshe, I venture to believe that another and much more rational explanation of these murmurs may be offered, as I will endeavour to show.

Dr. Walshe believes that systolic *basic* murmurs may arise from violent excitement of the heart, either through increased force of the current, or by derangement of its line of transit. This he has witnessed not only in hysterical females, but likewise in nervous male subjects. In hypertrophy with dilatation, systolic murmur may, according to him, be produced at the base, by misdirection of the current against the edges of the arterial orifices.



I have never met with an indubitable example of dynamic murmur at the base of the heart; all systolic murmurs in this situation, not dependent upon<sup>\*</sup> disease of the arterial valves or orifices, are, in my judgment, of hæmic origin; at least I have never met with any which could not on that assumption be satisfactorily explained.

In 1854 Dr. Stokes,\* whilst discussing the subject of functional murmurs, after referring to the opinions of Hope and Walshe who locate them exclusively in the arterial orifices, says: "But we must be cautious in rejecting the opinion that inorganic murmurs may be seated in the mitral orifice. I cannot help believing that I have observed cases of inorganic murmurs, which, so far as physical signs went, were closely similar to those of ordinary mitral disease with regurgitation into the auricles." He observed† prolonged first sound, passing into bellows murmur, in the space midway between the nipple and the left margin of the sternum, in continued and relapsing fever; in measles of asthenic character; and in typhoid variola. He quotes a case of maculated typhus, observed by Drs. Heslop and Lyons in the Meath Hospital, in which, after the first week of convalescence, the first sound of the heart was prolonged; and in the course of a relapse of brief duration, this prolongation passed into bellows murmur, which continued through a second relapse of ephemeral character, after which the patient, a young female, rapidly convalesced, with complete subsidence of abnormal cardiac signs.

In connexion with this case, Dr. Stokes remarks on the greater frequency of murmur of this character in typhoid than in typhus fever; and proceeds to observe, that "The prolongation of the first sound of the heart, whatever be its cause, is closely related to, though certainly different from, inorganic murmur. It is true that the same general causes seem to give rise to both." He adds: "It appears to arise from irregular contraction of the muscular fibrillæ. In some cases, especially in thin persons, we have found it attended with a peculiar vermicular sensation to the touch, very different from the fremitus of organic valvular

\* *Diseases of the Heart and Aorta.*

† *Opus citat.*, p. 428 and 502.

disease. Like the anæmic murmur it subsides during or after the convalescence of the patient, but I think that, when well established, its ultimate disappearance\* occurs much later than that of a true murmur."

A girl aged twelve, six days ill of simple non-maculated fever, exhibited prolonged first sound; on the ninth day, coincidently with increase of fever, and of the rate and force of the heart's action, this passed into bellows murmur, loudest between the left margin of the sternum and the nipple, but traceable, with progressively decreasing intensity, downwards towards the nipple, and upwards in the course of the aorta; it ceased when the patient sat up. On the eleventh day, with increase of weakness, the first sound and the murmur ceased to be audible, save faintly, behind the sternum. Stimulants were given; the action of the heart improved within an hour; and on the following day both first sound and murmur were again distinctly audible, the pulse being 92. Convalescence now set in; the pulse came down; the murmur gradually faded away; and at the end of a little more than a week, the patient was discharged quite well, the murmur having entirely ceased.\*

The preceding observations on the subject of dynamic murmurs possess a very high value, derived from the authority of the writer. They refer to examples of evanescent cardiac murmur, not basic, in association with asthenic fever, gradually disappearing with the progress of convalescence, and vanishing with restored health.

The murmur was, therefore, neither organic nor anæmic in origin.

To what cause, then, is it to be attributed? I hesitate not to answer, to parietal debility of the left ventricle. Of this, but in a minor degree, it furnishes evidence similar to that afforded  
\* by suppression of the first sound in low typhus, as noticed by Doctor Stokes.

Doctor M'Dowel has published† several cases of dilatation of the left ventricle, with consequent mitral incompetence and regurgitant murmur, without valvular disease.

\* *Dublin Quarterly Journal of Medical Science*, vol. xiv.

† *Opus citat.*

Doctor Bristowe has likewise published\* several such cases; but, in some of these there was, in addition to dilatation of the left ventricle, shortening and atrophy of the muscoli papillares and chordæ tendineæ, to which, in part, he attributes the mitral regurgitation and systolic apex-murmur. As causes of regurgitation, Bristowe denies the efficiency of anæmia, which "will not explain incompetence," and of fibrinous concretions; and he doubts the occurrence of dilatation of the auriculo-ventricular orifice to a degree sufficient to give rise to reflux. The murmur audible with the first sound and at the apex, in his cases, he attributes to "disproportion between the size of the ventricular cavities and the length of the muscoli papillares and chordæ tendineæ."

Doctor Hare detailed before the Pathological Society of London, a case in which he attributed mitral incompetence to lateral displacement of the points of origin of the muscoli papillares, and the consequent contraction of the latter in an altered direction.

Doctor Walshe, discussing the subject of mitral regurgitant murmur† says: "In rare instances it may be of dynamic mechanism."

Stark‡ is of opinion that in chlorosis, the fibres of the walls of the ventricles undergo relaxation, in consequence of the temporary derangement of nutrition which defect of red blood cells entails. From this results passive dilatation of the chambers of the heart, and, possibly also, relative inadequacy of the mitral valve.

Doctor Parrot§ holds that all murmurs designated anæmic, have their seat at the tricuspid, not the aortic orifice; and that a large proportion of murmurs arising in the course of acute rheumatism are produced in the former situation, as the result of debility and yielding of the walls of the right ventricle, of dilatation of the tricuspid orifice, and consequent inadequacy of the tricuspid valve.

\* *British and Foreign Med. Chirurg. Review*, vol. xx.

† *Opus citat.*, p. 100.

‡ *Gazette Hebdomadaire*, Paris, 1863; and *Archives Générales de Médecine*, 1866.

§ *Archives Générales de Médecine*, 1866, vol. xi.; and *Dublin Quarterly Journal of Medical Science*, November, 1869, p. 590.

Doctor Wilks\* believes that all mitral systolic murmurs associated with chorea are organic; and the result, not of rheumatism, but of the chorea itself. In all such cases he has found the endocardium in the vicinity of the mitral opening, studded with minute bead-like vegetations of fibrin, disposed in rows.

Da Costa† has met with non-organic mitral murmur in cases of obstructed pulmonary circulation; tubercular infiltration; asthma; pneumonia; and in a case of pleuritic effusion, in which, on examination of the body, the valves were found unaltered. He admits the possibility of temporary mitral regurgitation; but considers it not unlikely that many examples of mitral murmur are due to vibration of the blood, produced by increased tension of the mitral valve during temporary excitement of the heart, and favoured by a spanæmic state of the blood.

I cannot admit tension murmurs. I have never met with an example of such. The vibration theory, if correct, should apply to all cases of nervous palpitation in anæmic subjects, and vibration murmurs should be audible in all such cases. Yet how small the per-centage of these cases is, in which mitral murmur is actually present, all clinical observers can testify; and, as Dr. Cuming‡ justly remarks, the murmur when heard, if due to vibration of the blood, should be conducted by the current into the aorta and be there audible, which is notoriously not the case. The situation of maximum loudness of the murmur indicated by Doctor Da Costa, namely, the third intercostal space, is suggestive of its possible origin in the pulmonary artery; of such a murmur I shall, in the sequel, furnish some examples, and in subjects in whom the absence of organic lesion was legitimately presumable.

Doctor Da Costa is further of opinion, that functional valvular disorders may lead to organic valvular disease; the series being, derangement of the action of the heart, functional inadequacy of the valves, hypertrophy, organic valve-disease.

I fail to perceive how hypertrophy, as such, can lead to

\* *Medical Times and Gazette*, February, 1869.

† *American Journal of the Medical Sciences*, July, 1869.

‡ *Dublin Quarterly Journal of Medical Science*, November, 1869, p. 591.



organic disease of the valves. In simple hypertrophy, as exemplified in chronic renal disease, no such consequence as disease of the valves is ordinarily met with.

Bamberger is of opinion, that the so-called anæmic murmurs; those which occur occasionally in fever, and many of those which are developed in the course of rheumatism, are veritable mitral reflux murmurs; and that they are due to a weakened state of the papillary muscles, in consequence of which the valve is not rendered sufficiently tense, and after vibrations are permitted.

M. Raynaud\* contrasts non-organic and organic murmurs as follows: viz., "In the first (non-organic) the seat is at the base of the heart; it is propagated in the direction of the ascending aorta; it is soft and intermittent. The second (organic) is rougher; it assumes to some extent the character of the *bruit de scie*, *bruit de lime*, or *bruit de râpe*; it is more fixed, and, if it occupy the aortic orifice, the lesion is well pronounced, and possesses other characters."

It can now, however, be no longer maintained that the seat of non-organic murmur is exclusively basic. Nor, indeed, that murmurs of organic origin are invariably rough, and exhibit the characters above mentioned.

Doctor Peacock has given a list of cases, five in number, in which mitral regurgitation was announced by systolic murmur at the apex; but, in four of these cases, there was, in addition to dilatation of the mitral orifice, disease of the mitral valve in greater or less degree. In the fifth case there was adhesion of the pericardium, with left ventricular hypertrophy and dilatation, and enlargement of the mitral orifice, but "no material valvular disease."†

That non-organic mitral regurgitant murmur may arise from causes other than those above mentioned, I have, in another place,‡ endeavoured to show. I have given cases to illustrate the occurrence of such murmur in connexion with anæmia, purpura, masturbation, and excessive tobacco-smoking. To this

\* *Nouveau Dictionnaire de Médecine*, p. 388.

† *On Some of the Causes and Effects of Valvular Disease of the Heart*, 1865.

‡ *British Medical Journal*, November 16th and 23rd, 1867.

list I would now add chorea, and weak and fatty heart (see cases of J. O'B. and R. Burrows). It is quite true that in none of these cases, except those referred to under the last-mentioned two heads, has the negative diagnosis in relation to organic disease been confirmed by the evidence of *post mortem* examination of the body; because no opportunity for such was presented. But the cessation of the murmur under treatment, coincidently with the disappearance of irregularity in the heart's action, and the general improvement of the patient's health, constitute presumptive evidence amply sufficient to warrant the diagnosis given.

In the paper above referred to, I have expressed the opinion, "that an explanation of the occurrence of murmur under the circumstances now stated, must be sought mainly in physiological considerations having reference to the mode of combination, the direction, and the relative force of contraction of the *musculi papillares* of the left ventricle. That a yielding of a particular portion of the walls of the ventricle during the centripetal movement which takes place in the act of contraction, may so alter the direction in which one or both of the *musculi papillares* act upon the segments of the mitral valve, as totally to invert their function, by rendering them effective agents, not in closing, but, in *opening* the orifice of communication with the auricle, seems legitimately presumable from what occurs in the right ventricle, under similar circumstances, as shown by the late Mr. W. King.

"Such yielding may be due, not as in the right ventricle, to simple engorgement, but either to a want of sufficient contractile power in a particular part of the walls of the ventricle, or to ataxy or want of co-ordination in the contraction of the different portions of those walls.

"Without committing myself to a definite theory on this subject, I may state shortly the view which I entertain as to its pathology. The *musculi papillares* of the left ventricle (as also of the right) are so placed that in the state of complete ventricular systole they interlock, or pass to opposite sides of the chamber. In virtue of this interchange of position they so act upon the segments of the mitral valve, that the lines or

axes of their contraction decussate at an acute angle, and the valves are drawn, each towards the opposite side of the ventricle. Such a movement can take place manifestly only at the acme of ventricular systole, causing *active* closure of the valves, tension of these and of the chordæ tendineæ, and the sharp "click" constituting the terminal portion of the first sound. If from any cause whatever, involving inability of the ventricle to empty itself, such as yielding of a particular portion of its walls during their centripetal movement in the act of systole, the muscoli papillares are prevented from performing the movement of transposition already indicated, they will act upon the valves, *not* in the direction of the opposite wall of the ventricle, but towards that from which they take their origin, and will therefore divaricate the valves, and permit regurgitation.

"It has been objected that as, according to this view, the closure of the valves is effected only at the acme of systole, regurgitation may have taken place previously to its occurrence.

"But it should be remembered that the closure of the valves is passive, *quoad* the pressure of the mass of blood in the ventricle at the commencement of systole, and that their active closure is terminal, and rather an act of tension to prevent their being reversed at the acme of ventricular systole. If, at this moment, when the chordæ tendineæ are made tense by the contraction of the corresponding papillary muscles, one of them is made to act in the direction of the attached margin of the valve with which it is connected, owing to an irregular or centrifugal movement of a portion of the wall of the ventricle, the segment of the valve so acted upon is of necessity raised off the auriculo-ventricular opening, and regurgitation takes place at the close of systole.

"It is worthy of remark, as bearing upon this view, that regurgitant murmurs of this character are always, so far as I have observed, postsystolic, or occupy the terminal portion of the period of systole."

Finally I draw the following conclusions:

- "1. That regurgitation may take place at the mitral orifice independently of disease of the valves, and of dilatation of the orifice of the ventricle.

- “2. The murmur which announces this form of regurgitation is faint and abbreviated, usually confined to the area of the apex, and not associated with embarrassment of respiration, vascular turgescence, or the other usual signs of organic mitral regurgitation.
- “3. The pulse associated with this murmur is quick, abrupt, and feeble; and either rhythmically irregular, or of the alternately ascending and descending character previously described.
- “4. Examples of this murmur may be met with in those who are the subjects of anæmia, or purpura; or who have become enfeebled by masturbation, or by the excessive use of tobacco.”

To the list above given I would now add chorea, and fatty softening of the left ventricle, as previously stated.

With the experience I have since had, I incline to endorse the opinions thus expressed seven years ago, and am satisfied, whatever may be the value of the theory propounded, that causes, of which the above are examples, which debilitate the ventricular walls, may act indirectly as causes of mitral reflux of uncertain duration, but generally amenable to treatment.

Doctor Cuming of Belfast, has published,\* in a paper of great practical value, the particulars of two cases of similar import to the foregoing. In both, systolic murmur confined to the mitral area existed; and in one of these (case of R. C.) pulmonary and general vascular engorgement with anasarca were present; the patient died; there was no dilatation of the left ventricle, no atrophy of the muscoli papillares or chordæ tendinæ, and no disease of the mitral valve, or only a slight warty growth upon it quite inadequate to cause regurgitation. The muscular structure of the heart is reported as healthy; but, microscopic examination of it, which constitutes the only reliable evidence, negative or positive, on this subject, is not mentioned. This case is the first, as far as I am aware, in which mitral reflux murmur, accompanied by the usual symptoms of advanced organic disease; viz., pulmonary engorgement, hæmoptysis, and anasarca, existed; and in which dissection demonstrated the

\* *Dublin Quarterly Journal of Medical Science*, May, 1868.



absence of anomaly or disease in the cavities, walls, or valves of the heart. I have, since that date, met with two cases similar in their principal features to the foregoing, which, with the results of *post mortem* examination of the bodies, are given in the sequel (cases of J. O'B. and R. Burrows).

In Dr. Cuming's second case the patient had been weakened by purging; no antecedent murmur existed, as he had been examined and passed for a life assurance office. At the period of report, a soft bellows murmur of systolic rhythm and mitral diffusion existed; but it ceased after brief tonic treatment, coincidentally with restoration of perfect health.

This case presents many features of resemblance to those published by me in the preceding year, and to which Dr. Cuming refers. He, however, misapprehends the meaning I desired to convey when I stated that the absence of pulmonary congestion in my cases was, *pro tanto*, evidence of the absence of valvular disease. I did not, by any means, intend thereby to intimate that pulmonary and systemic engorgement, followed by the usual results, may not occur in such cases. I only wished to point out by the observation alluded to, the temporary character, and, inferentially, the non-organic cause of the murmur in my cases.

I am entirely of Dr. Cuming's opinion when he states that "it is very probable that several of Dr. Hayden's cases would have exhibited signs of pulmonary and systemic congestion had the regurgitation not yielded to treatment."

From what has preceded, I feel warranted in concluding that a murmur, postsystolic in time; in continuity with the first sound; extending to a variable length into the short pause; loudest at the apex, to which in most cases it is confined; loudest, likewise, in the recumbent posture, and diminished in intensity, or temporarily suspended, when the erect posture is assumed; unaccompanied by the symptoms and signs of pulmonary congestion, and by outward displacement of the apex-beat; and associated with rapid changes in the rate of cardiac and radial pulsation, irrespectively of physical exertion, indicates mitral reflux *without* structural lesion at the mitral orifice, or permanent dilatation of the left ventricle. I further hold, but not with equal confidence,

that in such a case the walls of the left ventricle would be found in a softened or atonic condition, and that to this, as previously explained, the murmur would be due.

The murmur is faint, soft, and blowing in character; and, in the instances which have come under my notice, has fallen short of the second sound; although it is conceivable that such should not invariably be the case. In the two examples above referred to, in which I had the advantage of a *post mortem* examination of the bodies, the result as regards the state of the valves was negative.

Doctor Nixon recently read before the Medical Society of the College of Physicians a paper on this subject, which was characterized by very considerable research and ability.\* He submitted the details of five cases, in three of which he had the advantage of examining the body after death. In all three the mitral valve was unaltered, and apparently adequate to close the orifice; but, in two of these, the muscular structure of the left ventricle was in a state of fatty degeneration. The third was an example of chronic meningitis and general mal-nutrition. Of the two cases in which death did not follow, one was that of a delicate female, who had been greatly reduced by flooding, during a miscarriage, eight months previously, and in whom both a basic and an apex-murmur existed, which ceased under treatment. The other was that of a young lady, who exhibited a murmur, only during nervous palpitation following mental disturbance or the use of green tea.

It will be observed, that in all these cases, except the last, there was general debility; and, in two of the number, the left ventricle was proved, by dissection, to be in a state of fatty softening. In the case of the young lady, nervous debility may be inferred.

Doctor Nixon is of opinion, that the conditions under which functional mitral murmur has been met with are so various, that no single explanation of the mode of its occurrence will suffice; but that, in most cases, it is caused by derangement of the consentaneous action which normally subsists between the muscular walls of the left ventricle and the papillary muscles of that

\* *Dublin Journal of Medical Science*, June, 1873.

chamber; and that this is due "to some defect in the vital power or condition of the heart itself, which leads either to atony of the papillary muscles, or derangement in the rhythm of their movements." I believe that in most cases, if not in all, debility of the walls of the left ventricle may be reasonably supposed to exist, and that the hypothesis which I have ventured to propound affords not only the most generally applicable, but the most rational explanation of the phenomenon.

Doctor Nixon's observations as to the differential diagnosis of this murmur are most judicious. The features upon which he relies, as of positive diagnostic value, may be briefly set forth as follows:

1. The murmur is intermittent, and variable as to intensity.
2. Usually present in the recumbent posture, it generally ceases when the patient sits up.
3. It is loudest, not at the apex, but over the body of the ventricle (Da Costa).
4. There is usually absence of pulmonary distress, and of accentuation and doubling of the second sound.
5. There is no alteration in the size or position of the heart.
6. The radial pulse is variable as to rate and volume.

The foregoing observations are of the utmost importance. I am not in a position to affirm all the rules just laid down for testing this murmur; but I have no hesitation in subscribing the majority of them, and in declaring that Dr. Nixon's paper is, in my judgment, the most valuable contribution hitherto made towards the differential diagnosis of functional murmur at the mitral orifice.

Skoda, writing of the diagnostic value of accentuated second sound in the pulmonary artery, as positive evidence of regurgitation at the mitral orifice in cases of systolic murmur at the apex, says:\* "If the force of this sound be not increased, the murmur then indicates that the surfaces of the valves, or the lining membrane of the ventricle about the arterial openings, are roughened."

Doctor Todd, likewise, held the opinion that a rough condition of the ventricular surface of the mitral valve was competent, of

\* *Opus citat.*, p. 229.

itself, to give rise to systolic murmur in the mitral area, liable to be confounded with that of mitral reflux.\*

He says: "But if the deposit take place on the *ventricular* surface of the valve (and it generally does so on the ventricular surface of its inner curtain) then you have no disturbance of valvular function. In both cases, however, you have a systolic bellows-sound, and in both cases that sound is best heard at the apex of the heart."

In the pseudo-murmur, however, the absence of Skoda's sign of accentuated second sound; the murmur being audible towards the base; and inaudible, or very faintly audible beneath the left scapula, suffice, in his opinion, to establish the distinction between the two cases.

Doctor Walshe also believes† that "vegetations on the ventricular surface of the (mitral) valve" are competent, without causing inadequacy or mal-adaptation of the valve, to produce systolic murmur at the apex.

The concurrence of so many eminent authorities in support or acceptance of the doctrine of systolic murmur of maximum intensity at the apex, due to simple roughening of the ventricular surface of the mitral valve, or of the adjacent portion of the ventricular wall, and liable to be confounded with that of mitral incompetency, would seem to constitute it an established dogma in cardiac diagnostics.

Nevertheless, I venture to question its soundness, and to express a doubt that it rests upon stronger evidence than *a priori* reasoning. At least, I have never fallen upon the details of a case, in which murmur of the site and rhythm above mentioned, viz., "systolic murmur best heard at the apex of the heart," was satisfactorily traceable to the cause now alleged, to the exclusion of all others competent to produce it. Nor have I met with a single example, in the living, which would warrant me in assigning to the alleged murmur, even provisionally, a place in cardiac pathology. I have, however, met with an example of *basic* systolic murmur due to roughening of the ventricular surface of the mitral valve.

\* *Clinical Lectures*, second edition, 1861, p. 77.

† *Opus citat.*, p. 99.



Doctor Todd lays too much stress, in regard to differential diagnosis, upon the fact that this murmur is heard but feebly, or not at all, at the left scapula; on the absence of intensified second sound; and on the less degree of disturbance of the heart and general distress which accompany it, as compared with the murmur of veritable mitral regurgitation.

I have repeatedly found indubitable mitral regurgitant murmur of organic origin, totally inaudible at the left scapula and unaccompanied by intensified second sound. Indeed, such is the rule in the case of soft murmurs of recent origin, and occurring in a feeble heart before appreciable derangement of the pulmonary circulation has supervened. As above shown, these negative signs and symptoms are characteristic of functional mitral murmur.

Dr. Todd indicates, however, the true distinction between systolic murmur at the apex due to simple roughening of the ventricular surface of the mitral valve, and the murmur of mitral reflux to whatever cause due, when he says that the former "may be heard well up to the base of the heart." Not only may it be so heard, but it is absolutely loudest at the right base, and audible throughout the arch of the aorta and its primary branches with great distinctness. If, with such a murmur, likewise, but less distinctly, audible at the apex, and unaccompanied by the other evidence of mitral regurgitation mentioned by Dr. Todd, the second sound in the aorta be unimpaired and free from murmur, the diagnosis of a systolic murmur of efflux in the aorta from roughening of the ventricular surface of the anterior mitral segment, or of the adjacent portion of the ventricle, may be positively made.

*Pseudo-murmurs* are sounds simulating cardiac murmurs, or liable to be mistaken for them. They arise from two sources; namely, pericarditis in the "dry" stage, and exocardial pleuritis.

The attrition-murmur of pericarditis, before it has assumed a character of decided harshness, may closely simulate veritable cardiac murmur. That is to say, in the early stages of the affection, when the free surfaces of the serous membrane, either by congestion of its vessels and arrest of its normal secretion, or

by fibrinous exudation as yet soft and plastic, cease to glide noiselessly over one another, as they do in a state of health, during the alternate movements of the heart in contraction and dilatation. The resemblance will be the stronger, and the diagnosis proportionately more difficult, when the attrition-sound is localized at the apex of the heart, as it occasionally is in the first instance. I have had, on two or three occasions, the opportunity afforded me of studying a pericardial friction-sound, limited to the apex, in its nascent stage (see case of John Clinton). Such murmurs are usually soft, and of single and systolic rhythm, during at least the first twenty-four hours of their existence, and at this stage are not readily distinguishable from regurgitant mitral murmurs. They are, however, more superficial in site, as indicated by their proximity to the ear of the observer, and less constant in character, undergoing a change in rhythm and in quality within the first two days.

The ordinary friction-sound of pericarditis is, however, of double rhythm, not strictly coincident in time with the sounds of the heart as alleged by all writers on this subject, with the exception of Dr. Walshe and Dr. Hyde Salter.\* The first attrition-sound coincides with the impulse and the first sound of the heart, and is therefore in the strictest sense systolic. The second, however, coincides, not with the second cardiac sound, but with the early portion of the succeeding or long pause, and is strictly postdiastolic in rhythm. I have met with not more than two exceptions to this rule, which may, therefore, be regarded as constituting an important element of distinction between the frottement of pericarditis and double aortic murmur. In the exceptional instances referred to, the second friction-sound was strictly diastolic, and, therefore, not by rhythm distinguishable from the bruit of aortic reflux.

Doctor Walshe says: "When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule; the friction-murmur may be very distinct during the postdiastolic silence, and occasionally falls between the first and second heart sounds. In point of fact, want of distinct synchronism of the

\* *Opus citat.*, p. 112, and *Lancet*, July 29th, 1871.

murmur with either of the two sounds is a very habitual and distinctive attribute.”\*

Sir T. Watson describes the acoustic phenomenon of pericarditis as a “to-and-fro” sound, as “of two surfaces rubbing backwards and forwards against one another.”† This is a just comparison, and a most accurate description of the sound.

Collin‡ compared it to the creaking of new leather, and named it, from the resemblance, *bruit de cuir neuf*. He says: “Perhaps this sound may be a constant symptom of pericarditis before the existence of effusion into the serous envelope of the heart, a symptom very fugacious in cases where this disease has terminated in a few days, of longer duration when it is chronic.” He adds: “I explain, then, this phenomenon by the rubbing of two layers of dried serous membrane. This kind of desiccation appears to be the first effect of inflammation upon membranous tissues, etc.”

Broussais says:§ “There is a phenomenon worthy of attention, to which, perhaps, it has not been given in a sufficient degree; this is the noise of parchment, which may be distinctly perceived by means of the stethoscope. Whilst exploring with this instrument in incipient pericarditis, one experiences a sensation such as that which might be yielded by two dry bodies, for example, of parchment, whilst rubbed one against the other; and this sign, when combined with the pain and anguish, should leave no doubt as to the existence of inflammation.”

It would thus seem that Broussais fully appreciated the diagnostic value of friction-sound as a sign of pericarditis.

Stokes, however, was the first writer who placed in a clear light the pathological significance of pericardial friction-sound.|| By a course of rigid observation of the vital phenomena of the disease in a number of typical and highly illustrative examples, followed by careful dissection of the bodies, he conclusively

\* *Opus citat.*, p. 112.

† *London Medical Gazette*, vol. xvi., part i., April 11th, 1835.

‡ *Les Diverses Méthodes d'Exploration de la Poitrine, etc.*, par V. Collin, Paris, 1824.

§ *Commentaires des Propositions de Pathologie*, 1829, tom. i., p. 398.

|| *Dublin Journal of Medicine and Chemical Science*, vol. iv., 1834; published September 1st, 1833.

established, not only the diagnostic value of friction-sound as a sign of pericarditis, but distinguished it by salient characteristics from other acoustic phenomena in the region of the precordium, of different origin and significance, with which it is liable to be confounded. As distinctive signs of this sound, he dwells upon the modification which it undergoes in consequence of the local or general abstraction of blood; its sudden supervention, variable character, and rapid change of situation; its occurrence with *both* sounds of the heart, where no sign of organic disease had previously existed; its subsidence under treatment, and not again becoming audible under excitement. He dwells likewise upon tactile vibration or *frémissement*, as distinguishing pericarditis from valvular lesion.

I cannot attach equal value to this last mentioned sign as distinctive of pericarditis. I have repeatedly observed *frémissement* of great intensity associated with valvular murmur exclusively, most frequently with that of presystolic rhythm, indicating obstruction at the mitral opening. No doubt, the fremitus of pericarditis is usually double, corresponding in that respect to the friction-sound; but pericardial murmur is occasionally single, and, when so, it is usually systolic, and located at the apex. In such a contingency fremitus might indicate, and numerically in about equal proportion, either constriction of the mitral orifice, or pericarditis at the apex. In a small percentage of cases it arises from mitral regurgitation. The mode of distinguishing these phenomena from one another will be pointed out further on.

Doctor Stokes very appropriately designates as "latent pericarditis" that form of the affection, of which the only evidence afforded consists in friction-sound, with or without *frémissement*.

Pericardial friction-sound is notably variable in character, in site, and in rhythm. It is likewise fleeting, or liable to temporary suppression.

The *quality* of the sound is in most instances harsh and unpleasing, and its *pitch* may vary, even in the same case, between the limits expressed by "grating" and "filing."

When these variations are presented in the same person, they



are noticeable from day to day, the changes being seldom more abrupt; and they are usually attained, by gradations, when the extremes above mentioned are reached, which, however, is not often the case.

The *site* of pericardial murmur is usually basic; but it may extend over the entire precordium, or it may be limited to the apex; and the relative frequency of the three sites would be indicated by the order in which they are above enumerated.

The existence of this murmur by preference at the base, is explained by the closer proximity of the visceral to the parietal pericardium in that situation, owing to the peculiar configuration of the heart. To this circumstance is likewise due the fact that, at the base, friction-sound is always of double rhythm. The transfer of the phenomenon from base to apex, or *vice versa*, is only apparent. Usually, the cases which present it most prominently, are those in which friction had been previously audible over the entire precordium; and in these the apparent transfer is due to suppression of the sound in one of the two situations mentioned. Occasionally, however, such is not the case, and a veritable extension of inflammation, to a previously unaffected portion of the pericardium, coincides with adhesion in the previous situation of friction-sound.

The rhythm of pericardial friction-sound depends upon the movements, not the sounds, of the heart. It is, therefore, as already stated, usually double at the base; because, in this situation, there are two periods in the cycle of cardiac movement when the visceral and parietal layers of the pericardium are in mutual contact; one, corresponding to ventricular systole, during which there is an upward movement with tension of the ventricles, and repletion with tension of the auricles; and the other coinciding with a downward movement or subsidence of the ventricles in diastole, and a simultaneous vermicular or vibratory contraction of the auricles. The first element of the double sound is therefore strictly systolic, sharp, and abrupt, because of the single and energetic character of the ventricular movement upon which it depends; whilst the second is postdiastolic, or posterior in time to the second sound of the heart; less loud, and of a prolonged and shuffling character, in accordance with

the weaker, more protracted, and somewhat undulatory movement to which it owes its origin.

Thus, a double pericardial friction-rhythm is recognised; namely, ventricular systolic, and ventricular diastolic; with the latter auricular systole coincides. The character of ventricular systolic frottement is uniform; not so, however, that of ventricular diastolic rhythm, because of its compound origin. The latter is usually composed of a succession of short and abrupt "rubs," commencing, at the base, *after* the second sound, but occasionally coinciding with it; and sometimes, though rarely, exhibiting by one of its elements, a distinct presystolic rhythm.

In my lectures on "Mitral Obstruction,"\* I urged the possibility of mistaking pericardial friction-sound, possessing this rhythm exclusively, for true endocardial presystolic murmur. But, inasmuch as such rhythm, even in combination, is rare in pericarditis, and still more so in the isolated form, the error of diagnosis adverted to is not likely to be committed.

In a very able lecture, published by the late Dr. Hyde Salter,† he described a pericardial friction-sound of triple rhythm; namely, ventricular diastolic, auricular systolic, and ventricular systolic. The second mentioned he truly alleges to be presystolic in time, and liable to be confounded with the endocardial murmur of mitral constriction. He gives, in illustration, the particulars of four cases, which may be summarized as follows:

Case 1. Triple frottement, which by-and-by became double by loss of one element (which, in particular, is not stated); auricular systolic friction-sound of presystolic rhythm existed at the inner part of the right third intercostal space; and, after death, this was found to depend upon a rough deposit of fibrin upon the right auricle. In the progress of this case, both elements of the double friction-sound at the base ceased simultaneously.

This is certainly not in agreement with my experience, which is to this effect, that double friction-sound at the base, in process of abolition, becomes single, for a brief period, by loss of one of its elements previously to final cessation. I cannot recall a single exception to this rule.

Case 2. Female; albuminuria; presystolic murmur over left

\* *Medical Press and Circular*, July, 1866.

† *Lancet*, July 29th, 1871,

third costal cartilage, one inch from the sternum, soft, single, and audible only within an area not larger than a halfcrown piece at the point indicated. On dissection, the left auricle only was found covered with rough lymph, and this coincided exactly with the seat of friction.

Case 3. A female; albuminuria and bronchitis, with cavity in right lung; frottement at inner part of right third intercostal space, loudest within an area of the size of a shilling, and audible over a space not larger than a crown piece. It was double; the first part rough and presystolic, the second fainter and softer, and systolic in rhythm. He concludes that the first was auricular systolic, and the second auricular diastolic. After a few days, friction became general over the heart. Autopsy: Recent general pericarditis, with effusion of soft lymph.

Case 4. A lady; sub-acute rheumatism; double frottement in right third intercostal space near the sternum, and limited in extent. The first was the louder of the two, and presystolic in rhythm.

Doctor Hyde Salter has demonstrated the truth of a theorem which I have long held to be demonstrable, but have never had the opportunity of proving; namely, that the rhythm of pericardial friction-sound is as the natural movement of the portion of the heart engaged, and the mobility of the opposed surfaces; and hence, with a knowledge of the movements of the different portions of the heart in health, a diagnosis of the seat and limitation of frottement may be made; and, conversely, that by accurate observation of the seat and rhythm of friction, the natural movements of the heart may be determined.

When the phenomenon is about to be suspended in consequence of liquid effusion into the pericardium, or to be suppressed by adhesion of its opposed surfaces, the friction-sound, even at the base, becomes irregular and, still later, single in rhythm (Case 13, Jane Armstrong).

At the apex a double friction-sound is rarely produced; and of this an explanation may probably be found in the peculiar character of the movements of this portion of the organ. In systole the apex moves upwards and forwards abruptly, and with much energy, gliding forcibly along the

anterior wall of the pericardium ; but, in diastole it recedes from the anterior wall, sinking, by its own weight, to the fundus of the pericardium ; hence, pericarditis at the apex is usually announced by a single and systolic friction-sound. The exceptions are of two kinds ; namely, where the posterior surface of the apex is also coated with lymph, and therefore competent to educe friction-sound in its descent. Lymph, however, is very rarely effused upon the posterior surface of the apex, and hence frottement of diastolic rhythm at the apex is of exceptional occurrence. Double *bruit de frottement*, of basic origin and great intensity, may be likewise audible at the apex by transmission ; but, its relative loudness at the apex and the base, respectively, or its point of maximum intensity, would, in such case, be a sufficient guide to the seat of its production.

Pericardial friction-sounds exhibit, in the majority of instances, what Dr. Stokes very appropriately designates as a "respiratory rhythm ;" that is, a rhythmical intensification corresponding to the acts of inspiration. This, he thinks, may be due either to descent of the diaphragm in inspiration, or to pleuritic friction at the lower and anterior portion of the left pleura. I cannot, however, admit either of these explanations as satisfactory. The cordiform tendon of the diaphragm, to which the pericardium is attached, is all but motionless in respiration, as shown by the unaltered position of the apex during the fullest inspiratory effort ; and the seat of greatest intensity of the inspiratory frottement is not the lower, but the upper portion of the precordium. The rhythmical intensification of pericardial friction-sound, corresponding to inspiration, is due, I doubt not, to the advance of the anterior edge of the lungs between the chest wall and the pericardium during inspiration, by which the opposed surfaces of that membrane are brought into more forcible contact. If, from any cause whatever, as, for example, pleuritic adhesion, this advance of the lungs should not take place, I venture to assert that in such a case the pericardial friction-sound would be devoid of inspiratory rhythm.

The area of diffusion of these sounds is usually circumscribed, or nearly so, by the limits of the precordium ; a characteristic by



which it is distinguished from all other acoustic phenomena of cardiac origin. In children it is usually audible over the entire front of the chest; and also, but less frequently, to a corresponding extent in the back, owing to the high conducting properties of the thorax at this period of life. It attains its maximum of intensity, and is most extensively diffused, when pericarditis supervenes upon ventricular hypertrophy (see case of Clinton).

In females, owing to the same cause as in children, Dr. Stokes surmises friction-sound may be likewise more extensively audible. I cannot say that I have been able to confirm this observation. In advanced age, and in gouty subjects, the chest walls, rendered more resonant by ossification of the costal cartilages, and by partial ankylosis of its articulations, may likewise serve as better conducting media for sound; especially that having the character of frottement, which involves perceptible vibration.

The *cause* of pericardial friction-sound is regarded by the great majority of writers on this subject, as consisting in plastic fibrinous deposit upon one or both surfaces of the pericardium, generally both. Collin, however, held that it was due, exclusively, to the mutual friction of the opposed pericardial surfaces in a state of dryness from arrest of the normal serous secretion. His explanation of the phenomenon is given at page 291.

Hope advocated a similar view, but less exclusively and dogmatically. He was of opinion "that deficient lubricity of the pericardium from defective secretion in the earliest stage of inflammation, may possibly be *one* of the causes of the creaking sound, independent of lymph."\*

*Cessation* of pericardial frottement may be due to cohesion of the opposed serous surfaces; or, to serous, sero-sanguineous, or purulent effusion separating them. Under the influence of these several causes, the suspension or suppression of the pseudo-murmur is gradual, and in the process its rhythm and force are much modified. If adhesion take place, the friction-sound is permanently suppressed in the seat and to the extent of adhesion; and

\* *A Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1839, p. 168.

its cessation above, whilst it continues to be audible at the lower portion of the precordium, is conclusive evidence of progressive adhesion, commencing superiorly. An inversion of this order, however, would not be of similar import, because the cessation of friction-sound progressively from below upwards might be due, either to adhesion or liquid effusion; and the differential diagnosis would rest upon other evidence, mainly that yielded by percussion.

The friction-sound of pericarditis is liable to be confounded with:

1. Double aortic murmur.
2. Single (systolic) aortic murmur.
3. Systolic mitral murmur.
4. Left pleuritic frottement with short and rapid breathing.
5. Left pericardial pleuritic frottement, synchronous with the impulse of the heart.

The features of resemblance between double valvular murmur at the aortic orifice, and to-and-fro pericardial friction-sound at the base, are, the reduplication of murmur in both alike, the alleged synchronism of the murmurs with the normal sounds of the heart, and the maximum intensity of both at the base.

The aortic murmurs, however, are transmitted upwards in the course of the aorta, and, of these, the systolic likewise into the carotid and subclavian arteries; they fade *gradually* away, and equally in all directions except in the line of the aorta, as the stethoscope is shifted from the point of their greatest intensity. They are strictly synchronous with the sounds of the heart in the great majority of cases; they do not vary in rhythm, and, though loud, they are manifestly not of superficial origin; they are not modified by ordinary treatment, nor are they intensified by pressure made upon the precordium.

Pericardial friction-sounds, on the contrary, abruptly terminate at the limits of the precordium, except in children and in cases of great enlargement of the heart; they are not audible to a greater distance in one direction than another; they are never transmitted into the neck; they are not both synchronous with the sounds of the heart, the second being, as already stated, post-diastolic in time; they are peculiarly variable as to rhythm,

quality, intensity, and situation; superficial as to origin; much intensified by surface pressure; and readily modified by treatment. Finally, they most frequently exhibit an inspiratory rhythm, or periodic intensification corresponding to the inspiratory act;\* and are usually associated with *frémissement*. This latter is a most valuable indication, though not pathognomonic of pericardial friction. It is much more frequently met with in association with the latter than with valvular lesion; indeed, the friction-sound of pericarditis is never unaccompanied by fremitus, save in its nascent stage, and in that immediately preceding its final extinction, when it is soft and faint.

*Frémissement cataire* is one of the most remarkable phenomena exhibited by the heart in a state of disease. It has been aptly compared by Laennec, to the sensation communicated to the open hand placed upon a cat's back whilst the animal purs; and it usually coincides in rhythm and varies in intensity with the murmurs audible beneath, the harsher murmurs always yielding the strongest tactile vibration. It may be most readily perceived by means of that portion of the palm corresponding to the roots of the fingers, placed with moderate pressure over the seat of friction.

I have found *frémissement* associated, not only with pericardial attrition-sound, but likewise with valvular murmur of every kind, except those of single and diastolic rhythm at the orifice of the aorta, or the pulmonary artery. In both these cases I conclude that arterial systole, though capable of producing a murmur of reflux, is incompetent to develop a force sufficient to educe thoracic vibration.

I have already intimated that, exceptionally, the friction-sound of pericarditis may be diffused extensively, and even universally, over the chest, in children, and likewise in adults who are the subjects of cardiac hypertrophy. In the latter cases I have heard

\* This sign, which is due to the pressure of the pericardium against the anterior surface of the heart by the advancing edges of the inflated lungs, is, when present, pathognomonic of pericarditis; but it fails where vesicular emphysema of the edges, or great enlargement of the heart exists; because, in the former case, the edges of the lungs are permanently inflated and overlie the pericardium; and in the latter they are permanently displaced outwards, and cannot advance during inspiration from want of space. Permanent adhesion of the lungs to the sides of the pericardium will likewise neutralize this sign.

it beneath both clavicles, on both sides of the back, and along the dorsal spine. In children loud valvular murmurs are no less extensively diffused. Nevertheless, attention to the preceding distinctions, as well as to those of a general kind, to be hereafter mentioned, having reference to the pulse, the seat of apex-pulsation, and the area of precordial dulness, by which double valvular disease at the aortic orifice is characterized, will suffice to establish the differential diagnosis between murmurs due to that disease, and the pseudo-murmurs resulting from pericarditis.

Aortic murmur of single and systolic rhythm is liable to be confounded with bruit de frottement, only when the latter approaches extinction by cohesion of the opposed surfaces. At this period the friction-sound of pericarditis is usually, even at the base, single and systolic in rhythm, less loud and harsh than previously, and, therefore, more closely similar in quality to the murmur of valvular obstruction. The latter, however, is *always* propagated in the line of the ascending aorta, and transmitted into the carotids; the former *never*.

From the murmur of mitral reflux, pericardial friction-sound of single and systolic rhythm and apex origin, may be distinguished, chiefly, by the positive signs which characterize the former; namely, its being transmitted into the axilla, audible, in most cases, at the inferior angle of the left scapula, and associated, after the initial period, with intensification of the second cardiac sound in the pulmonary artery, together with the signs of pulmonary congestion, and engorgement of the right side of the heart.

Indeed, by no other positive characteristics than its unsteadiness as to quality, site, and rhythm, and its brief duration, can single systolic friction-sound at the apex be distinguished from mitral reflux murmur; but these, in conjunction with the above given character of mitral reflux murmur, will justify a confident diagnosis, even in the most obscure cases, after careful examination once or oftener repeated.

From the respiratory frottement of pleuritis of the left side, the friction-sound of pericarditis may be readily distinguished, by causing the patient to suspend respiration for a moment. The procedure will, of course, likewise suspend the friction-



sound, if it be of pleuritic origin ; not so, however, if its seat be the pericardium.

The distinction between the attrition-sound of pericarditis, and that of pleuritis engaging the pericardial portion of the pleura and adjacent edge of the lung, and deriving its rhythm from the movements of the heart, not from those of the lung, is attended with the utmost difficulty.

Doctor Stokes\* states that in the latter case, the murmur is loudest rather outside than within the limits of the precordium. I can confirm the accuracy of this distinction.

Doctor Mayne,† discussing the positive value of frottement synchronous with the action of the heart, as a sign of pericarditis, inquires: "But even in simple pleuritis may not the mechanical impulse of the heart against the lung, when covered by inflamed pleura, produce a distinct frottement synchronous with the systole of that organ, and thus cause difficulty in the diagnosis, unless general symptoms are taken into the account?" To this I answer, that in such a case friction-sound would be confined to the apex, and either inaudible at the right base and in the middle line, or audible in these situations only by transmission.

In the course of pericarditis, a systolic bellows-murmur may be developed at the base or at the apex of the heart.

Doctor Stokes would regard the former as due to co-existent endocarditis, and the latter as the result of weakening of the heart.

Doctor Walshe conceives it possible that a basic systolic murmur may result from constriction of the roots of the aorta and pulmonary artery by exuded fibrin ; and he quotes the opinion of Martin-Solon, to the effect that a murmur of this rhythm and site may arise in the stage of serous effusion, from liquid pressure upon the roots of these vessels.

I doubt much the occurrence of murmur from either of the two last mentioned causes. I have never heard a basic murmur in the absence of valvular engagement, or of strong collateral evidence that it was of hæmic origin. On the other hand, I

\* *Dublin Journal*, vol. iv., 1834.

† *Dublin Journal of Medical and Chemical Science*, vol. vii., May 1st, 1835.

have repeatedly found the roots of the great vessels enveloped in organized fibrin, and the pericardium distended with serum, when no such murmur existed.

I am satisfied that the cause assigned by Dr. Stokes for temporary apex-murmur, occurring in this connexion, is correct. I more than doubt the alleged resolution of valvular inflammation, in a degree sufficient to restore the normal character of the sounds of the heart, where murmur due to inflammatory exudation had previously existed ; and I have already expressed the opinion, supported by clinical evidence, that initial regurgitant murmur may result from simple debility and yielding of the walls of the left ventricle, under the centrifugal pressure of the contained blood. Such a condition of the heart, involving no structural disease, would be, of course, eminently curable ; and the murmur arising from it, of temporary duration, and readily suppressed by tonic treatment.

## CHAPTER IV.

### CAUSES OF DISEASE OF THE HEART.

THE *causes* of disease of the heart, including that of its fibrous envelope, are manifold, but may be conveniently classified under three heads; namely, hæmic, mechanical, and nutritive.

HÆMIC CAUSES ...	{	Rheumatism.
		Chronic renal disease.
		Scarlatina.
		Measles.
		Small-Pox.
		Septicæmia.
		Puerperal fever.
		Phlebitis.
		Scurvy.
		Purpura.
		Cancer.
MECHANICAL CAUSES	{	Tuberculosis.
		Syphilis.
		Blows or falls upon the chest ; or wounds of the pericardium or heart.
	{	Constriction of the chest, with laborious exercise.
		Malformation or deformity of the chest, with pressure upon the heart.

NUTRITIVE CAUSES are those which, by inducing malnutrition, lead to structural degeneration of the heart.

Hæmic causes of disease of the heart are such as operate through the blood. They consist in the presence of certain irritant principles in the circulation; whether primarily introduced from without, as in the case of the exanthematous poisons; the result of imperfect assimilation, as lactic acid; of failure of elimination, as urea; or defect of constitution, as in scurvy. These causes operate, in the first instance, as chemical irritants, and, secondarily, through the changes so produced, as mechanical

excitants of hypernutrition, with the single exception of renal disease, the influence of which upon the heart as a morbid agent, would seem to be exclusively mechanical.

The proclivity of persons suffering from rheumatism, especially in the acute form, to inflammatory affections of the heart, has been known from the earliest period of medicine. Doctor Latham\* states that two-thirds of the cases of acute rheumatism treated by him in St. Bartholomew's Hospital, during the five years from 1836 to 1840, both included, suffered likewise from inflammatory affections of the heart.

This is certainly in excess of the usual proportion, according to the experience of others; and Dr. Latham, whilst admitting that such is the case, suggests as an explanation the great assiduity of his clinical clerks in providing "interesting cases" for his wards. Doctor Sibson† records thirty-four cases of acute cardiac affection occurring in seventy-four of acute and subacute rheumatism; *i.e.*, very nearly one-half of the total number. This would approximately represent the proportion in which rheumatism has been complicated by cardiac disease in my own experience. The form which this complication is prone to assume is that of inflammation of the lining or of the investing membrane of the heart; namely, endocarditis, or pericarditis. Of the sum total of cardiac complications of rheumatism recorded by Latham, three-fourths were endocarditis, one-twelfth pericarditis, and one-seventh consisted of the two-fold affection. Of Sibson's cases, four-fifths were endocarditis, one-tenth, pericarditis, and one-tenth, peri- and endocarditis combined.

As regards sex and age. In Latham's returns the proportion of males and females affected with cardiac complication was about equal; and Sibson states that nearly one-half of his cases (32 out of 74) were under twenty years of age.

Returns made for childhood (under twelve years) would, I have no doubt, show a still larger proportion of rheumatic cases complicated with acute inflammation of the heart; indeed, the occurrence of acute rheumatism without this complication in childhood is the exception.

\* *Diseases of the Heart*, vol. i., 1845, p. 144.

† *British Medical Journal*, August 13th, 1870.



Doctors Rilliet and Barthez\* report that, of the children under their care affected with rheumatism, more than one-third suffered from pericarditis: namely, 4 in 11.

The following statistics, borrowed from the able and exhaustive treatise of Dr. Fuller,† are of especial value in relation to this portion of our subject. It will be seen that they deal, not only with the gross proportion of cardiac disease of rheumatic origin, but likewise with the relative prevalence of its different forms, as influenced by age and sex, in acute, subacute, and chronic rheumatism respectively.

ABSTRACT OF CASES OF ACUTE AND SUBACUTE RHEUMATISM, ADMITTED INTO ST. GEORGE'S HOSPITAL, BETWEEN THE 1ST OF JANUARY, 1845, AND THE 1ST OF MAY, 1848.

Age of Patients.	Cases of Acute and Subacute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exocardial Disease.	Valvular Disease of uncertain Date.
Under 15	22	12	3	5	3	—	1
15 to 20	82	49	4	11	26	4	4
20—25	92	49	3	6	26	9	5
25—30	79	41	1	4	22	9	5
30—35	40	14	1	1	4	6	2
35—40	28	12	—	1	4	6	1
40—45	15	5	1	—	2	2	—
45—50	14	4	—	—	2	2	—
50—55	3	—	—	—	—	—	—
55—60	4	1	—	—	—	1	—
	379	187	13	28	89	39	18

Of the 13 cases of pericarditis, 6 occurred in men, and 7 in women; or one in every 12·4 men, and one in every 6·7 women.

Endocardial affection 89, viz., 48 in men, or one in every 3·4, and 41 in women, or one in every 2·6.

\* *Traité Clinique et Pratique des Maladies des Enfants*, vol. i., p. 210, quoted by Dr. Fuller.

† *On Rheumatism, Rheumatic Gout, and Sciatica*, third edition, 1860, c. ix., p. 257 et sequent.

ABSTRACT OF CASES OF ACUTE RHEUMATISM ADMITTED INTO ST. GEORGE'S HOSPITAL,  
DURING THE SAME PERIOD.

Age of Patients.	Cases of Acute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exocardial Disease.	Valvular Disease of uncertain Date.
Under 15	15	9	2	5	2	—	—
15—20	58	37	4	10	21	1	4
20—25	63	39	3	6	22	5	—
25—30	50	33	1	4	20	4	4
30—35	22	11	1	1	4	4	1
35—40	17	8	—	1	3	3	1
40—45	9	4	1	—	1	2	—
45—50	9	3	—	—	2	1	—
50—55	1	—	—	—	—	—	—
55—60	2	1	—	—	—	1	—
	246	145	12	27	75	21	10

Of the 12 cases of pericarditis, 5 occurred in men, or in the proportion of one in 7·7; and 7 in women, or one in 5·2.

Of recent endocardial affection, 39 occurred in men, or one in every 2·4; and 36 in women, or one in every 2·1.

ABSTRACT OF CASES OF SUBACUTE RHEUMATISM ADMITTED INTO ST. GEORGE'S  
HOSPITAL, DURING THE ABOVE MENTIONED PERIOD.

Age of Patients.	Cases of Subacute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exocardial Disease.	Valvular Disease of uncertain Date.
Under 15	7	3	1	—	1	—	1
15 to 20	24	12	—	1	5	3	3
20—25	29	10	—	—	4	4	2
25—30	29	8	—	—	2	5	1
30—35	18	3	—	—	—	2	1
35—40	11	4	—	—	1	3	—
40—45	6	1	—	—	1	—	—
45—50	5	1	—	—	—	1	—
50—55	2	—	—	—	—	—	—
55—60	2	—	—	—	—	—	—
	133	42	1	1	14	18	8

Of the 14 cases of recent endocardial affection, 9 occurred in men, or one in every 8·3, and 5 in women, or one in 5·1.

Pericarditis occurred in one out of every 92 men, and in one of every 41 women; or in little more than one per cent. among males, and one-half per cent. among females.

Thus it would appear that about one-half the cases of acute and subacute rheumatism registered, presented some form of cardiac affection, or 1 in every 2·02; and some form of recent cardiac affection in more than one-third of the total number, or 1 in every 2·7; and finally, some cardiac affection of long standing in more than one out of every eight cases, or 1 in 7·9.

Of the total of 130 cases of recent cardiac affection, 114 occurred in persons affected with acute rheumatism; and of those affected, nearly one-half, or 1 in every 2·06, exhibited recent heart-disease of some form.

Cardiac affection, in one of its many forms, occurred in about one-third (1 in every 3·1) of the cases of subacute rheumatism, and *recent* cardiac affection of some kind in about one out of every six and a-half cases, or 1 in every 6·6.

In the following table the preceding results are compared with those obtained by other observers.

	Total of recent Car- diac Affec- tion.	Cases of Acute Rheumatism.	Proportion in which recent Affection of the Heart occurred.
Cases in St. George's Hospital	119	246	1 in every 2·06
Dr. William Budd's Cases ..	21	43	1 „ 2·04
M. Bouillaud's Cases ...	65	114	1 „ 1·75
Dr. Latham's Cases ...	74	136	1 „ 1·83
Dr. John Taylor's Cases ...	27·5	49	1 „ 1·78
Total ...	306·5	588	1 „ 1·91

With regard to the numerical proportion of cases of acute rheumatism complicated with pericarditis and endocarditis respectively, Doctor Fuller states that the former occurred in about one-sixth, and the latter, including the twofold affection, endo-pericarditis, in one out of every 2·3, of the cases collected by him.

The following Tables, borrowed from Dr. Fuller's work, exhibit in a connected form the absolute and the relative proportions of the two forms of cardiac affection in acute rheumatism, according to the experience of different observers.

## PERICARDITIS.

	Pericarditis.	Acute Rheumatism.	Proportion in which Pericarditis occurred.
Cases treated in St. George's Hospital ...	39	246	1 in every 6·3
Dr. Basham's Cases ...	14	66	1 „ 4·7
Dr. William Budd's Cases ...	5	43	1 „ 8·6
Dr. Latham's Cases ...	22	136	1 „ 6·1
Dr. Macleod's Cases ...	54	307	1 „ 5·7
Dr. Taylor's Cases ...	8	49	1 „ 6·1
Total ...	142	847	1 „ 5·97

## ENDOCARDITIS.

	Endocarditis.	Acute Rheumatism.	Proportion in which Endocarditis occurred.
Cases treated in St. George's Hospital ...	107 0	246	1 in every 2·29
Dr. W. Budd's Cases ...	17·3	43	1 „ 2·48
Dr. Latham's Cases ..	65·0	136	1 „ 2·09
Dr. Taylor's Cases ...	25·4	49	1 „ 1·92
Total ...	214·7	474	1 „ 2·25



## RELATIVE FREQUENCY OF RECENT EXOCARDIAL AND ENDOCARDIAL AFFECTION.

	Cases of Pericarditis.	Cases of Endocarditis.	Relative proportion of Pericarditis and Endo- carditis.
Cases treated in St. George's Hospital ...	39	107.0	1 to every 2.7
Dr. William Budd's Cases ...	5	17.3	1 „ 3.4
Dr. Latham's Cases ...	22	65.0	1 „ 2.9
Dr. Taylor's Cases ...	8	25.4	1 „ 3.1
Total ...	74	214.7	1 „ 2.9

Doctor Fuller remarks: "The great excess of endocardial affection exhibited in these tables, arises, I believe, not from the greater frequency of endocarditis, but from the large number of cases in which a murmur is occasioned either by purely functional causes, or by temporary imperfect closure of the mitral orifice consequent on irregular contraction\* of the structures connected with the valves, or by the presence of fibrin deposited on the valves, without the concurrence of endocardial inflammation. I believe somewhat less than one-third of all recent cardiac murmurs met with among patients suffering from acute rheumatism\* will be found to result from pericarditis, and somewhat more than one-third from endocarditis, whilst the remainder will be referable to one of the three causes above specified as contributing to the production of valvular murmur." He adds, that out of 107 cases in which endocardial murmur existed, 23 left hospital quite free from it.

Bamberger, as quoted by Niemeyer,\* states that endocarditis occurs in acute articular rheumatism in the proportion of about twenty per cent.; pericarditis fourteen per cent.; myocarditis in much smaller proportion; and that the tendency to inflammation of the heart and pericardium is greater in proportion to the number of joints engaged.

I believe endocardial murmur may, and frequently does, arise from the first and the last mentioned cause in the course of acute rheumatism; but I have never met with any satisfactory

\* *Text-Book of Practical Medicine*, 1869, vol. ii., p. 481.

evidence, either clinical or necroscopic, in support of the doctrine of systolic patency of the auriculo-ventricular orifices from irregular or spasmodic contraction of the papillary muscles. I believe, further, that by far the greater number of these evanescent murmurs are due to temporary weakness or atony of the ventricular walls.

Age is a potent element in the causation of rheumatic inflammation of the heart. As shown in the preceding tables, 114 out of 130 examples of recent cardiac affection occurred in persons under 30 years of age; and of 41 examples of pericarditis, 23 occurred in persons under the age of 20, and 37 in patients under the age of 30, leaving 4 only to be apportioned amongst those of all ages above 30. Thus it would seem that rheumatic pericarditis is essentially a disease of early life.

In acute rheumatism I have found pericarditis to exceed endocarditis in frequency; and where the latter existed, the former was likewise present with very few exceptions. Males have been more frequently affected with cardiac complication of rheumatism than females, and the period of accession of the cardiac affection varied from the second to the twenty-seventh day, from the date of first illness. The friction-sound has been of every conceivable rhythm and site; from double systolic (case of Mary N.) at base, to prediastolic at apex (case of Mary K.). Presystolic friction-sound at apex I have repeatedly met with (see cases of L. O'B. and J. D.); it is interesting chiefly with reference to differential diagnosis from the presystolic murmur of mitral constriction. The complications of rheumatic pericarditis which I have met with, have been pleuritis (single and double), myocarditis, and endocarditis.

The murmur of acute rheumatic endocarditis is, with very few exceptions, mitral as to origin, and systolic as to rhythm. Once established it is usually coeval with the life of the patient. My experience, in regard to this matter, does not by any means coincide with that of Dr. Sibson, who discharged one-half of his cases of endocarditis "well and free from cardiac valve-murmur;"\* nor with that of Dr. Latham, seventeen of whose cases, out of a total of sixty-three, or nearly one-fourth, left hospital

\* *British Medical Journal*, August 13th, 1870.

free from murmur.\* I have not had the good fortune to meet with more than one such case out of some hundreds; that is, complete cure of endocarditis, in the sense of final cessation of a previously well pronounced endocardial murmur; which, as regards endocarditis, is the *only* evidence of cure I am willing to admit.

Doctor Sibson has correctly stated† that the earliest announcement of valvular engagement in acute endocarditis, consists in prolongation of the first sound (cases of Jane A. and Mary N.), which, in the course of a few days, is gradually transformed into veritable murmur. I have had, however, no experience of the inverted order of these phenomena which has so often fallen under the cognizance of Dr. Sibson; namely, the transition of murmur into prolonged sound, as a preliminary step to its final extinction.

The portion of the civil population most liable to cardiac disease are precisely those who are most exposed to the causes of rheumatism, and, pre-eminently, to sudden changes of temperature with insufficient clothing, depressed spirits, and deteriorated health. Tailors and milliners, especially, are examples of the truth of these remarks. After long confinement in their miserable workshops, overheated by gas, ill-ventilated, and with insufficient cubic space for the numbers crowded into them, these poor people are exposed to cold in travelling to, or actually whilst resident in, their humble and ill-provided lodgings. Their food is unwholesome or insufficient, their clothing light, and their minds depressed by the unsuccessful struggle for existence. Rheumatism, of a very acute character, and complicated with cardiac inflammation in a proportion higher than the average, is consequently of common occurrence amongst them.

The agent by which, in rheumatism, valvular inflammation is produced, is generally considered to be lactic acid. Dr. Richardson has shown, experimentally,‡ that this agent introduced within the peritoneum of a living animal, is capable of exciting acute inflammation of the endocardium, manifested chiefly upon the valves.

\* *Opus citat.*, p. 147.

† *Loco citat.*

‡ *The Asclepiad.*

Doctor Stokes remarks,\* with truth, that cardiac and articular inflammation are, alike, only accidents of the rheumatic fever; and that either, or both at the same time, may complicate it. He mentions two examples of rheumatic pericarditis, in one of which, observed by himself, the symptoms of pericardial inflammation preceded the implication of the joints by a period of ten days; and in the other, which occurred in the practice of Dr. Graves and was declared by symptoms and signs, the interval preceding arthritis was five days. In reference to rheumatic pericarditis he has arrived at the following conclusions; viz.,

1. "That though the combination of pericarditis is common, yet that the disease of the heart is more closely related to the rheumatic fever than to the inflammation of the joints.
2. "That the liability to pericarditis is in direct proportion to the violence and duration of the fever.
3. "That in the apyrexial cases of acute arthritis, the liability to cardiac inflammation is but slight.
4. "That pericarditis may be developed at any period of the disease, and even precede the arthritis.
5. "That every variety and degree of pericarditis may occur in connexion with acute rheumatism, from the simple, dry, latent pericarditis, to the worst forms, combined with inflammation of the endocardium and muscular structure."

He notices three forms of rheumatic pericarditis: viz., 1st, that in which the disease, as regards symptoms, is latent, physical signs alone declaring its existence; 2ndly, that which is announced by a train of symptoms recently set up, and dependent upon copious liquid effusion; and, 3rdly, that in which symptoms of cardiac distress precede the physical signs by one or two days.

As regards the relative frequency of acute inflammation of the pericardium and endocardium, single and combined, he has come to the conclusion that it may be represented by the following series:

1. Acute pericarditis, with endocarditis.
2. Acute pericarditis, without endocarditis.
3. Endocarditis, without pericarditis.

\* *The Diseases of the Heart and Aorta*, 1854, p. 46, et sequent.



Guided by what I have personally witnessed, I believe that the foregoing scale correctly represents the relative numerical proportion in which acute pericarditis and endocarditis are met with in actual practice.

There are several sources of fallacy which, if not carefully avoided, may lead to an opposite conclusion. These will be found fully discussed in connexion with the subject of endocarditis. Here it is only necessary to state that, in my opinion, acute primary pericarditis is of more frequent occurrence than acute primary endocarditis. I do not, however, coincide with Dr. Stokes in the opinion that acute pericarditis is more frequently met with in combination with acute endocarditis, than in the isolated form. Indeed, I hold that the compound disease should be placed, not at the head, but at the foot of the scale.

The occurrence of hypertrophy of the heart, as a sequel of chronic renal disease, was first noticed by Dr. Bright. After narrating one out of many cases illustrative of this connexion, he remarks: "What influence the state of the heart exerts over the kidney is less obvious, nor is it clear that it is at all instrumental in promoting the mottling (granular disease) of the organ, though in many cases the two organs are found simultaneously diseased."\*

From this extract it is evident that Bright, even at this early date, had perceived a relationship between renal and cardiac disease, although he did not rightly interpret the connexion. Subsequently, however, he exhibited a clearer insight into the mutual relationship of these two diseases. After pointing out the connexion of hypertrophy of the left ventricle with renal disease, he says: "The obvious structural changes in the heart have consisted chiefly of hypertrophy with or without valvular disease, and what is most striking, out of fifty-two cases of hypertrophy, no valvular disease whatever could be detected in thirty-four; still, however, leaving twenty-two without any probable organic cause for the marked hypertrophy generally affecting the left ventricle." In explanation of the pathological relationship he assumes either a direct irritant action of the blood upon the heart, or that it "so affects the minute and

\* *Reports of Medical Cases*, 1827, vol. ii., part i., p. 240.

capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."\* Thus, to Bright is clearly due, not only the discovery of the connexion between left ventricular hypertrophy and chronic renal disease, but likewise that of the causative relationship in which the latter stands towards the former affection, and the correct theory of its operation, namely, minute arterial and capillary resistance.

Doctor George Johnson supplemented and confirmed this theory,† by showing that in all the forms of chronic renal affection included under the designation of Bright's disease, the muscular coats of the minute renal arteries are hypertrophied; and further, that hypertrophy of the systemic arteries exists in these diseases.‡ Finally, he offers a connected view of his theory in a serial form as follows; viz., blood contamination by retained excreta, capillary resistance, hypertrophy of the minute arteries, and hypertrophy of the left ventricle.§

Jaccoud|| accepts the mechanical theory of Traube in explanation of hypertrophy of the heart in Bright's disease generally, but he does not consider it of universal application. In many cases he regards it, with Bright, as due to the altered state of the blood.

According to Dr. Grainger Stewart,¶ cardiac hypertrophy was present in 40 per cent. of his cases of the "inflammatory form" of Bright's disease, and was apparently referable solely to the kidney disease. The proportion in which hypertrophy existed increased with the stage of the disease thus:—

1st stage, 12 per cent. of the fatal cases.

2nd stage, 38 per cent.

3rd stage, 100 per cent.

In the "amyloid" form he met with cardiac hypertrophy only in the proportion of 4 per cent. of the cases, and in all of these the renal disease was in its third or atrophic stage.

In the "cirrhotic" form of the disease 46 per cent. of the cases

\* *Guy's Hospital Reports*, vol. i., 1836.

† *Med. Chirurg. Transactions*, vol. xxxiii.

‡ *Lancet*, December 21st, 1867.

§ Clinical Lecture, *British Medical Journal*, March 7th, 1868.

|| Clinical Lectures on Medicine, delivered at the Charité Hospital, Paris, 1867.

¶ *Bright's Diseases*, 1868.

presented enlargement of the heart, which was attributable to the renal disease exclusively; whilst the proportion in which cardiac hypertrophy, with valvular or vascular disease, was associated with cirrhotic disease of the kidneys, was still larger.

Amongst the hæmic causes of disease of the heart I regard the poison of scarlatina as next to rheumatism in frequency, and perhaps superior to it in potency. It attacks the endocardium by preference, seldom engaging the pericardium, and more frequently the mitral than the aortic valve.

Many of the most formidable examples of valvular lesion that I have met with owed their origin to scarlatina. The patients are generally children, and rarely survive the second period of life. The complication is usually declared in the second week of the fever, but occasionally in the first week, or in the stage of desquamation.

Trousseau maintains that the frequency of cardiac complication in scarlatina is due, not to that disease, but to the co-existence of rheumatism, which, as he alleges, is a not unusual concomitant of scarlatina, occurring in the proportion of one-third of the cases in adults, and manifested by pains in the joints from the third to the eighth day. Under these circumstances, endo- and pericarditis frequently occur in the stage of decline of the scarlatina, the former somewhat more frequently than the latter.\*

In the course of measles and small-pox likewise, but less frequently than in scarlatina, from the operation of the specific poison of these diseases, endocarditis is set up, and followed, in time, by the usual structural alterations of the heart. In the low, asthenic, or typhoid form of small-pox, however, pericarditis, latent, or declared only by physical signs, is the usual cardiac complication. Such is likewise the case in diffuse inflammation, puerperal fever, and phlebitis, as long since pointed out by Dr. Stokes.

Scorbutus and purpura occasionally give rise to pericarditis of the hæmorrhagic form, or may complicate an ordinary pericarditis by determining extravasation of blood into the pericardium.

\* *Clinical Medicine*, Sydenham Society's edition, 1868, vol. i., p. 397.

This subject will be discussed further on in connexion with that of pericarditis.

Cancerous and tuberculous pericarditis are also of occasional, but very rare, occurrence.

Ricord describes morbid changes of the heart and other organs, which he compares to syphilitic tumors or nodes.\*

Amongst the *mechanical* causes of disease of the heart, blows or falls upon the chest hold a prominent place; they usually act injuriously upon the organ by causing rupture of the chordæ tendineæ or semilunar valves, or detachment of the latter from the walls of the vessels.

Wounds of the heart are very rarely the cause of chronic disease or functional impairment of the organ; they are usually followed either by proximate death, or ultimate and complete recovery. Extravasation of blood into the pericardium, consequent upon a wound, may, however, give rise to pericarditis and its results.

Constriction of the chest, as by tight lacing or bracing, especially if conjoined with laborious exercise, is eminently conducive to cardiac hypertrophy, and disease of the arch of the aorta, from extra tension. It is in this way that the excessive mortality from disease of the heart and aorta in the British army is to be accounted for.

Doctor Robertson has shown† for the four years ending 1866, a mean ratio of mortality from disease of the heart of 40 per 1,000 in the army, and 12 per 1,000 in the navy. This formidable mortality in the army from cardiac disease, he attributes, in an especial manner, to the severe drill to which the young recruit is subjected whilst tightly braced up in a close-fitting jacket and high stock. The "running drill," in particular, he regards as most pernicious in its operation; and he sustains this opinion by reference to the number of admissions into hospital for cardiac disease amongst the Royal Marine Artillery, during the two years preceding, and those immediately succeeding, the introduction of that form of drill into the corps. In the year

\* *Clinique Iconograph, de l'Hôpital des Vénériens.*

† "On Diseases of the Heart and Aneurism in the Royal Navy and Army," by G. Robertson, M.D., *Medical Press and Circular*, July 7th, 1869.



1865 the number was 24, and in 1866 it was 26. In the summer of 1867 the running drill was introduced, and the admissions for cardiac disease the year after its introduction amounted to 47, in an average force of 1,750 men. During the first quarter of the succeeding year the number was 25. This drill is practised only during the winter months; and despite the severe drill this body of men undergo habitually in the summer, he observes that during that season the number of admissions for disease of the heart has been much below that registered in the winter.

The proportionate excess of cardiac disease in the navy, Dr. Robertson inclines to attribute to rheumatism, generated by the twofold cause of vicissitudes of temperature and the constant habit of washing the decks.

Assistant-Surgeon Myers\* treats the subject of excessive prevalence of cardiac disease in the army with much ability; and, with the view of exhibiting this in contrast with the ratio amongst corresponding classes of the civil population, he shows the proportion of rejections for disease of the heart amongst recruits for the years 1864, '65 and '66, respectively, to have been 19·78, 22·22, and 25·13 per 1,000.

Inspector-General Lawson, as quoted by him, gives the general mortality from disease of the circulating system, including aneurism and cardiac disease, in the civil population and the army, respectively, between the ages of 15 and 44 years, as follows:

Civil population	..	..	..	·48 per 1,000
Army	...	...	...	·84 „

and, excluding aneurism,

Civil population	...	...	...	·45 per 1,000
Army	...	...	...	·50 „

But even the preceding table does not fairly represent the relative mortality; because soldiers invalided for disease of the heart are placed to the account of the civil community, and their deaths are registered against it. Thence arises a twofold

\* *Prize Essay on the Etiology and Prevalence of Diseases of the Heart among Soldiers*, 1870.

derangement, namely, reduction in the ratio of fatal cardiac disease in the army, and undue elevation of it amongst civilians.

Mr. Myers further gives, as the result of his own inquiries, the following figures, representing the relative mortality from disease of the heart amongst civilians and soldiers of corresponding ages :

			Army.	Civilians.
20 to 24 years ...	...	...	·12	·25
25 and upwards	...	...	1·56	·94

Thus, it would seem that within the age of 20 to 24 years the death-rate from this cause is lower in the army than amongst civilians; but from 25 years upwards it is much higher. No doubt soldiers, who are admitted into the service at or about the age of 20 *on account* of their good health, contrast favourably in this respect with an equal number of civilians who are *not* selected with reference to this condition; and at the age of 24, these youths have not been as yet subjected to the various causes, which confer upon the military service of Great Britain the distinction of a preponderating mortality from disease of the heart, for a sufficient length of time to develop their full operation.

For the purpose of exhibiting the relative death-rate and invaliding from cardiac disease in the naval and military services, Mr. Myers submits the following statistics, given in summary, for the period 1860 to 1865 inclusive :

	Died	Ratio per 1,000 of admitted.	Invalided.	Ratio per 1,000 of admitted.
Army	259	·90	1·506	5·26
Navy	228	·66	1·184	3·44

With reference to the special forms of disease of the circulatory system found to prevail in the army, the annexed Tables, borrowed from Mr. Myers's work, are of much value :

RELATIVE PROPORTION OF DEATHS AND INVALIDING IN THE HOME SERVICES, OWING TO THE VARIOUS DISEASES OF THE CIRCULATORY SYSTEM, IN THEIR ORDER OF FREQUENCY, DURING THE FIVE YEARS, 1863 TO 1867.

Diseases of the Circulatory System.	Deaths.	Ratio per 1,000 of Total Deaths.
Aneurism ... ..	138	431·23
Morbus Valvularum Cordis ... ..	103	321·87
Hypertrophia Cordis ... ..	21	65·62
Degeneratio Cordis ... ..	21	65·62
Pericarditis ... ..	21	65·62
Morbus Cordis (doubtful) ... ..	5	15·62
Carditis .. ...	4	12·5
Atrophia Cordis ... ..	3	9·37
Atheroma Arteriosum ... ..	2	6·25
Varix ... ..	1	3·12
Syncope ... ..	1	3·12
	320	...

Diseases of the Circulatory System.	Invalided.	Ratio per 1,000 of Total Invalided.
Morbus Valvularum Cordis .. ...	518	369·98
Varix ... ..	473	357·41
Hypertrophia Cordis ... ..	201	149·26
Morbus Cordis ... ..	37	27·48
Aneurism ... ..	35	26·
Carditis ... ..	29	21·54
Palpitatio ... ..	21	15·6
Pericarditis ... ..	17	12·63
Angina Pectoris ... ..	6	4·45
Atrophia Cordis ... ..	4	2·97
Syncope . ... ..	3	2·22
Degeneratio Cordis ... ..	2	1·48
	1,346	...

As regards aneurism, the difference observable in the foregoing tables would seem due to difficulty of diagnosis.

But it may be said that from rheumatism, which operates so injuriously amongst the civil population as a cause of cardiac disease, the soldier is not exempt; nay, it may even be assumed that in the camp and bivouac he is more exposed to the causes of rheumatism and, its frequent consequence, organic disease of the heart, than persons of corresponding classes engaged in civil

avocations. But, exposure of this kind occurs only in the time of war, and is, therefore, exceptional. Again, there is no evidence whatever that rheumatism is more prevalent in the army than amongst the civil population; whereas morbus cordis notoriously is so.

Doctor Maclean has shown\* that out of one hundred and fifty-one patients treated for heart-disease in Netley Hospital, six only had suffered from acute rheumatism.

After rheumatism, the most frequent cause of morbus cordis is renal disease; this, however, operates chiefly after the period of life when the soldier retires from active service. The same may be said of degenerative structural changes in the heart.

Amongst seventy patients suffering from cardiac disease at Netley, Dr. Parkes was able to collect the histories of only two or three who had been the subjects of rheumatic, renal, syphilitic, or other affections, of a character adequate to account for the supervention of disease of the heart.

In India, for the years 1865, 1866, and 1867, the invaliding on the score of heart-disease was only in the proportion of 3·88 per 1,000. Hence, residence in that country cannot be adduced as a special cause of the prevalence of heart-disease amongst soldiers. The excessive mortality and invaliding from this cause in the army, must, therefore, be charged to the account of some agency, to the operation of which the military service is peculiarly exposed.

Doctor Maclean says:† “The labouring men have their clothes perfectly free, so do also sportsmen and tourists; but the soldier is compelled to do his work under the utmost possible disadvantages, as regards the weight he has to carry, the mode of carrying it, and the entire arrangement of his dress and equipment. Many men fall out in a state of extreme distress, and many surgeons assure me that nothing but a strong feeling of *esprit de corps* prevents many more from doing so.”

Mr. Myers writes:‡ “In civil life, when any prolonged or violent exertion of the body has to be made, be it for pleasure or

\* *Army Medical Report*, 1867.

† *Journal of the United Service Institution*.

‡ *Opus citat.*, p. 32.



otherwise, the great point first attended to is to remove all the various constrictions of ordinary dress, it being well known by all classes of society how necessary it is, under such circumstances, to allow the chest its fullest powers of expansion, and consequently the greatest possible freedom to the heart and lungs.

"Now, the method of equipping the soldier is directly opposed to this. His tunic is made to fit as tightly as the skill of the tailor can accomplish, any defect in this respect being probably corrected, after the careful scrutiny of the colonel or adjutant, by a little paring, and finally, by the addition of a padded waist-coat, which the soldier takes upon himself to supply.

"His waist-belt adds to the constriction below the chest, and his tunic collar above it (with or without the stock), and then, to complete the artificial chest-case, the knapsack straps supply all that is requisite, whilst the pouch-belt adds its share to the general compression.

"The chest thus fixed, as it were, in a vice, has little or no power of expansion, and the circulation through heart, lungs, and great vessels is proportionately impeded."

Mr. Myers adduces the following extract from the report of the Government Committee, appointed in 1864 to inquire into this subject, as confirmatory of the opinion urged by himself in the preceding passages.

"During exertion, the movements of the chest increase greatly; deeper breathings are made, the diameter of the chest enlarges in all directions, causing greater expansion of the lungs; the blood flows much more rapidly, and the changes in it, and the evolution of carbonic acid are trebled and quadrupled in amount, and the heart acts much more quickly and forcibly. If anything destroys the equilibrium between the powerful action of the heart, and the capacity of the lungs to receive the blood propelled into them by the heart, the necessary consequence is an accumulation of blood in the cavities and walls of the heart, which leads to an imperfect action of that organ, and to organic changes in its cavities and walls." He adds: "When experiments were made at Chatham to ascertain the comparative amount of distress caused by the accoutrements of various

European armies, it was unquestionably proved that, though all produced it more or less, ours ranked first in prejudicial effect."

It would appear, as indeed might have been anticipated, that next after hypertrophy of the left ventricle, the form which organic disease of the heart most commonly assumes in the army is that affecting the aortic valves. Thus, as shown by the *post mortem* returns of Netley Hospital from October, 1860, to November, 1869, the numerical proportion of the several valvular lesions stood thus; viz., aortic valve disease, 50; mitral valve disease, 25; aortic and mitral valve disease, 22.\*

Doctor Maclean's returns show a less striking disproportion; thus: aortic valve disease, 72; mitral valve disease, 54; total cases, 126.†

Barclay, Chambers, and Ormerod show the following relative proportion between these several forms of valvular lesion in civil life; viz.: aortic valve disease, 192; mitral valve disease, 205; aortic and mitral valve disease, 259; total cases, 673.‡

Inspector-General Lawson showed that deaths from aortic aneurism were eleven times more numerous in the army than in the male civil population;§ and Dr. Maclean states: "Between April 1st, 1867, and April 1st, 1869, thirty-six cases of aneurism of the aorta were under treatment in the clinical wards of Netley," and in these there was evidence of "a rheumatic history in five, and a distinct syphilitic history in only three of the cases."||

The excessive use of alcohol has been charged with the preponderance of aneurism in the army; but on apparently insufficient evidence.

The difficulties opposed to the circulation of the blood through the arch of the aorta by the constriction of the chest, due to the soldier's dress, would seem to afford a more rational explanation. Syphilis, though, unfortunately, very prevalent in the army, is likewise common amongst the civil population of towns; and the difference in its relative prevalence in the two classes men-

\* *Opus citat.*, p. 57.

† *Army Medical Report*, 1867.

‡ *British and Foreign Med. Chirurg. Review*, vol. xiv.

§ *Army Medical Report*, 1866.

|| *Ibid.*, 1867.

tioned, is by no means proportionate to that which represents the disparity in the per centage of aneurism amongst them. I believe the conclusion is inevitable, that the greater number of these cases is due "to mechanical obstruction to the circulation in the soldier when he is undergoing exertion, caused by the general constriction of his neck and chest by faulty clothing and accoutrements."\* If this conclusion be legitimate, it must follow that, in the soldier, the arch of the aorta is most frequently the seat of aneurism; and that such is actually the case the following statistics adduced by Mr. Myers would seem to prove: ascending aorta, 37; arch, 38; descending, 12; thoracic, 7; abdominal, 15; total number of cases of aortic aneurism, 109.†

It behoves every humane man who has been made sensible, by the evidence of the statistics now adduced, of the great and preventable sacrifice of life imposed upon the rank and file of the British army by an error of the War Office, to raise his voice against it, and to urge the officials at the head of this department to endeavour, at length, to comprehend the elements of a problem which everybody who has thought over the matter, except themselves, would seem to have long since mastered. This official blindness seems the more unaccountable, since the information by which the public mind has been enlightened on this subject has been supplied by the Army Medical Reports exclusively. The independence and public spirit exhibited by the medical officers by whom these reports have been compiled, is most creditable to the Army Medical Service, and cannot fail to be ultimately productive of reform in the dress and accoutrements of the soldier.‡

Deformity of the chest, more especially that known as "pigeon-chest," by interfering with the movements of the heart, becomes a cause of mechanical irritation, and, ultimately, of hypertrophy of the organ. Incurvation of the spine, whether antero-posterior or lateral, by inducing contraction of the chest in one or more of its diameters, operates in a similar manner.

\* Mr. Myers, *Opus citat.*, p. 70.

† *Opus citat.*, p. 72.

‡ Since the above was written we are glad to learn that certain improvements have been made in this respect.

Hence, in all such cases the action of the heart is quick and tumultuous. At first the sounds are sharp and clear, as in simple functional excitement ; but, after some time they become masked, owing to the supervention of hypertrophy from persistent and excessive functional activity.

In a similar manner, hypertrophy of the heart may be, and frequently is, induced by artificial constriction of the chest, as exemplified in the vicious habit of tight lacing. Than this habit, so prevalent amongst young females of the affluent classes, no example can be adduced more strikingly illustrative of the practical evils arising from ignorance of the elements of physiology. If ladies were instructed in the first principles of animal mechanics, the laws which govern respiration and circulation, and the penalties attached to their infringement, it is not likely, despite the inexorable requirements of fashionable society, that they would wilfully provoke the terrible consequences of early disease of the heart.

Diseases of degeneration of the heart are due to two several causes ; namely, direct failure of nutrition, and interstitial exudation. To the former cause are attributable softening, attenuation and fatty degeneration of the organ, and also calcification of adventitious deposits in or upon it ; and to the latter, congestive hypertrophy, induration, and atrophy of tissue.



## CHAPTER V.

### DISEASES OF THE HEART.

DISEASES of the heart are divisible into two great classes; viz.,

1. Organic.
2. Functional.

*Organic* diseases comprise four groups; namely, those engaging:

The investing membranes,  
The parietes,  
The valves, and  
The nutrient vessels of the heart.

*Functional* diseases of the heart are of two kinds; viz.,  
Muscular asthenia, and  
Neurosis.

*Pericarditis*, or inflammation of the pericardium, is presented under one of two forms; namely, the acute and the chronic.

*Acute pericarditis* is usually an accompaniment or a complication of acute or subacute rheumatism; it may, however, be the only well pronounced rheumatic manifestation. The disease is most frequently declared in the course, and within the first week, of an attack of articular rheumatism. Usually the articular pain is either greatly mitigated, or even entirely suspended, on the supervention of pericarditis. Acute pericarditis may, however, be the result of wounds or other mechanical injuries of the pericardium, of the irruption of pus, as from an hepatic abscess; or of the exudation or leakage of blood into the pericardium, as in the hypnotic state. It may likewise arise as a consequence of ulceration of the heart or pericardium; from exposure of the body, whilst heated, to the influence of cold; from retrocession of gout or erysipelas, as stated by Mr. Adams; or in the course of an exanthem. Phlebitis, or pyæmia, and the rapid cure of psoriasis by local applications (Wood), have been likewise adduced as causes of acute pericarditis.

Fuller states that it complicates rheumatism under ordinary treatment once in 5·97 cases.\* He has met with six cases in which pericarditis occurred from two to seven days before articular inflammation, and he holds that these two affections exercise no reciprocal influence, the one upon the other. I cannot help thinking, however, that in this opinion he is mistaken. I rather agree with Dr. Sibson,† who maintains that inflammation of the joints does exercise a very potent influence, by reflex irritation, in determining the occurrence of pericarditis.

Three stages of the disease have been usually distinguished, as clearly stated by Dr. Mayne;‡ viz., 1st, that of suspended secretion of serum, and consequent dryness of the pericardium; the only pathological change, with the exception of its opposite, serous effusion, recognized by Collin;§ 2nd, effusion of lymph or fibrin; and, 3rd, effusion of serum.

Doctor Mayne was of opinion that the first of these stages was declared only by general symptoms; viz., oppression of breathing, rapid pulse (130 to 140), anxious expression of face, etc. He stated that, in his experience, it repeatedly preceded friction-sound by thirty hours, as inferred from the existence, during this period, of the symptoms above mentioned.

Graves states that excited and irregular action of the heart often *precedes* the signs of pericarditis; and Stokes has observed the symptoms of pericarditis to precede friction-sound by two days.

Mayne was of opinion that the pericardium in the state of simple dryness of its surface, is incapable of yielding an audible attrition-sound.

Collin, on the contrary, held that this is actually the condition of the pericardium indicated by the *bruit de cuir neuf*.

Stokes doubts, but does not deny, the competency of simple dryness of the pericardium to produce *frottement*.||

\* *Diseases of the Chest*, 1862, p. 153, *et sequent*.

† Address in Medicine. British Medical Association. *Pamphlet*, 1870, p. 3.

‡ *Dublin Journal of Medical and Chemical Science*, vol. vii., May 1st, 1835.

§ *Commentaires des Propositions de Pathologie*, 1829, tom. i, p. 398.

|| *Dublin Journal of Medical and Chemical Science*, vol. iv., September 1st, 1833.

In his work (*Diseases of the Heart and Aorta*, p. 9) published more than twenty years later (1854), Dr. Stokes admits, with scarcely any qualification, the occurrence

Doctor Walshe holds that a state of dryness, with vascular engorgement of the free surface of the pericardium, is competent to yield friction-sound of a grazing, or even of a scraping character, during the movements of the heart.\*

I have never met with a case which would warrant me in asserting, that a state of simple dryness and vascularity of surface may give rise in the pericardium to veritable friction-sound. I do not, however, deny the possibility of an occurrence, which, theoretically, would seem not improbable. In every instance, without exception, in which I have had the advantage of determining, by *post mortem* examination of the body, the condition of the serous surface of the pericardium, where friction-sound of indubitable pericardial origin had existed during the patient's last illness, I have found lymph in greater or less quantity effused upon that surface; and out of a great number of such examples, I cannot recall one which afforded the slightest warrant for the assumption that the lymph so effused was in process of removal by absorption, notwithstanding the positive assertion to this effect made by many writers of eminence.

If it is possible, as Dr. Mayne held,† and in this opinion I quite coincide with him, to predict from general and special symptoms the proximate advent of pericardial *frottement*, the pericardium must exhibit, concurrently with the manifestation of these symptoms, the earliest pathological change which characterizes a serous surface in process of inflammation, namely, capillary injection and suspension of secretion. In two of Dr. Mayne's cases the diagnosis of pericarditis was made from symptoms alone, thirty hours before *frottement* was perceptible.

I have repeatedly, in the course of rheumatic fever, anticipated the advent of pericardial *frottement*, by at least twenty-four hours, from the sense of precordial oppression experienced by the patient, the accelerated breathing and pulse, anxious

of pericardial *frottement* in the first stage of pericarditis, from simple dryness of the serous surface; and Dr. Hope (*opus citat.*, p. 168) thinks that "deficient lubricity of the pericardium, from defective secretion in the earliest stage of inflammation, may possibly be one of the causes of the creaking sound, independent of lymph."

\* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 216.

† *Loco citat.*

expression of countenance, and, especially, from the abrupt, energetic, and tumultuous action of the heart, associated with a peculiar rustling sound; other acute inflammations of the chest having been excluded. In all the above examples, I believe that the serous surface of the pericardium was already vascular and dry; yet, by the most careful examination, friction-sound was not then detectable. Within the course of the succeeding twenty-four hours, however, indubitable attrition-murmur became distinctly audible, and converted the *presumptive* into a *positive* diagnosis of pericarditis. Without the confirmatory evidence afforded by friction-sound, it is manifest that a positive diagnosis of pericarditis could not have been made in the cases adverted to, because the symptoms, taken individually or collectively, might have been due to other causes, however strong the probability of their dependence upon pericarditis. Da Costa declares that pericarditis can be recognized only by means of physical signs, adding, there are no general symptoms that prove a pericarditis to exist, not even that of the so-called characteristic precordial pain.\* Corvisart, and even Laennec himself, strange as it may seem, were unacquainted with the physical signs of pericarditis; their diagnosis of this affection rested upon presumptive evidence alone. Bertin, Broussais, and Andral laboured under the same disadvantage, notwithstanding that the discovery of the pathognomonic sign of pericarditis, the *bruit de cuir neuf*, was published by Collin the same year (1824) in which Bertin's book appeared, but some months earlier, as shown by the disparaging allusion to it contained in that work, two years before the second edition of the celebrated *Traité de l'Auscultation Médiate*, and five years anterior to the works of Andral and Broussais. The latter, indeed, whilst fully admitting the diagnostic value of the sign discovered by Collin, gives no evidence that he had made much use of it in practice. He says:—"But there is a phenomenon worthy of more attention than it has, as yet, perhaps received. It is the sound of parchment, which one perceives distinctly by means of the stethoscope.

"In exploring with this instrument, in cases of incipient pericarditis, one experiences the sensation yielded by two dry bodies,

\* *Medical Diagnosis*, third edition, 1870, p. 355.



such as parchment, rubbing against one another; and this sign, when associated with pain and anguish, can leave no doubt as to the existence of inflammation. There is no reason why we should not profit by this observation for the purpose of making a precise diagnosis of the disease," etc.\*

Still more singular is it that Laennec, who has made such free and profitable use of the stethoscope in the diagnosis of pulmonary and cardiac disease, should have failed to recognise its application in that of pericarditis, and not have noted the striking and pathognomonic phenomenon of friction-sound in connexion with that disease. Collin was no stranger to Laennec, as, in the preface to the second edition of his great work, the illustrious discoverer of mediate auscultation proclaims his indebtedness to that distinguished observer for much and valuable assistance rendered in the collection of his clinical records.

Doctor Latham was one of the first physicians who recognised the diagnostic value of pericardial *bruit*. He says:—"I am not called upon to determine the general value of auscultation, as an aid to diagnosis. Probably it does not deserve all the high commendation of its inventor and its early advocates, and still less the absolute contempt and rejection which it has incurred at the hands of others. I confine my remarks to a single sign derived from it, contributing something, I believe, towards the diagnosis of a particular disease, and am content to affirm, that during more than three years in which I have practised the method of auscultation with some diligence, and, as a security against self-deception, have admitted no result of my own observation which has not been confirmed by that of others, the sign in question, of the peculiar sound accompanying the contraction of the ventricles, has not been absent in any one authentic case of pericarditis. And in three years the number of such cases in so large an hospital as St. Bartholomew's is considerable. My observation is restricted to *rheumatic pericarditis*. The same sign may attend pericarditis arising under other circumstances, but I do not know that it does.

"In rheumatic pericarditis the *brouissement*, or *bruit de souff-*

\* *Loco citat.*

flet, is always among the earliest symptoms referable to the heart, and sometimes the very first.”\*

From the preceding extract it will be seen that, whilst identifying and correctly associating the acoustic sign of pericarditis, Dr. Latham failed to appreciate its precise pathological significance, and the full extent of its diagnostic application. Nevertheless, he is entitled to distinguished notice in the history of this subject.

To Dr. Stokes belongs the merit of having been the first not only to assign their full diagnostic value to the acoustic phenomena of pericarditis, but to lay down precise rules, not even now diminished in value, for their identification.†

In his valuable and original paper, six cases of pericarditis are narrated, which may be briefly summarised as follows:—

Case 1. Loud friction-sound and *frémissement*, due to a second and recent attack of pericarditis, were perceived. Old adhesions and much semi-cartilaginous deposit were found on *post mortem* examination of the body.

Case 2. Double friction-sound, but varying in quality at different points of the pericardium, and not audible beyond one and a-half inch from the precordium, whilst the normal sounds of the heart were extensively audible. *Frottement* persisted at one point in the precordial region, whilst elsewhere it had ceased. The diagnosis of adhesion of the pericardium at all points, save that just mentioned, was made, and on *post mortem* inspection of the body, was found to be correct. This was an example of “latent” pericarditis; *i.e.*, in which the presence of the disease was announced by physical signs exclusively.

Case 3. Latent and dry pericarditis. Friction-sound varied in quality in different situations. Diagnosis confirmed by autopsy.

Case 4. A child, aged five years. Double friction-sound audible under both clavicles, and along the spine. Extensive precordial dulness, and epigastric tenderness. On examination of the body after death, dry pericarditis, and considerable hypertrophy of the heart, with slight recent thickening of the mitral and aortic valves, were found.

\* *London Medical Gazette*, vol. iii., January 17th, 1829, p. 214.

† *Dublin Journal of Medical and Chemical Science*, vol. iv., September 1st, 1833.

Case 5. Double friction-sound, which underwent change of quality and situation in the progress of the case, and exhibited, moreover, a respiratory rhythm. *Post mortem* examination not obtained.

Case 6. Double friction-sound which underwent change of character and site, and on the supervention of precordial dulness, ceased to be audible. Dulness subsequently cleared up, and friction-sound became again audible, and in the course of convalescence assumed the character of *bruit de soufflet*. Recovery.

These cases were of the utmost value to the science of medicine at the time they were published, because they settled affirmatively, once and for ever, the question of the practicability of diagnosing pericarditis actually in progress, by means of physical evidence exclusively. But, furthermore, from the observation of these cases, rules were constructed for the differential diagnosis of pericarditis, which may safely challenge comparison with anything since written on this subject.

In an especial manner are the variations in the intensity, quality, and rhythm of friction-sound in different situations within the precordium; and its modifications from day to day, and under treatment, insisted upon, as distinctive of this phenomenon.

In the progress of this work I shall have occasion again to refer to this important contribution to medical literature.

Doctor (now Sir Thomas) Watson followed, at a short interval, with a valuable paper\* confirmatory of Dr. Stokes' opinion as to the unique character and special diagnostic value of attrition-sound in pericarditis; contributing cases in illustration, one of which, as an example of latent pericarditis, was of especial value at this period, when the possibility of the diagnosis of that disease from acoustic and tactile evidence exclusively was still called in question. The diagnosis was confirmed by *post mortem* examination of the body.

In the same year Dr. Mayne contributed a memoir of the utmost value on this subject.† His cases, which are numerous,

\* *London Medical Gazette*, vol. xvi., part i., April 11th, 1835.

† *Dublin Journal of Medical and Chemical Science*, vol. vii., May 1st, 1835.

would alone be sufficient to establish the independent physical diagnosis of pericarditis. Yet, he declares it as his opinion, that physical signs are not of themselves sufficient in all cases; adding, "In some cases this sign (friction-sound) will not be evident at any one period of the entire disease, and in others it may readily be mistaken for similar sounds which are characteristic of other organic lesions." The differential diagnosis from endocarditis is, he says, in many instances attended with the greatest difficulty; especially is this the case when pericarditis has supervened upon organic disease, the stethoscopic phenomena being due exclusively to the latter.

I cannot agree in the opinion that pericarditis may pass through its ordinary stages without giving rise to friction-sound at any period of the disease. Of course, I do not mean to deny that the phenomenon may not have actually come under the cognizance of the attendant physician, from causes having no reference to its existence; such as the stage of the disease at which examination may have been made, the rapidity with which serous effusion had taken place, etc. But the development of *frottement*, cognizable to the senses, within a period, it may be very brief, subsequent to the first stage of the disease, I hold to be a necessary pathological sequence of ordinary inflammation of the pericardium. I further believe that in the absence of this sign, the diagnosis of pericarditis can be only presumptive; and that for the purpose of diagnosis, either physical signs are insufficient *only* when they are ill-pronounced or entirely absent at the time of examination.

That pericardial friction may be readily mistaken for other phenomena I freely admit; but this in no measure diminishes its value as a specific sign; it should rather induce a more careful study of its distinctive peculiarities, which, by such means, will be found not only cognizable, but amply sufficient to establish the identity of pericarditis where they exist.

Pericarditis may occur singly, or with intrinsic or extrinsic complications. Thus:



- |   |   |
|---|---|
| 1. SIMPLE PERICARDITIS ... ..                             | Idiopathic (?)                              |
|   | (a) Endo-pericarditis.                      |
|   | (b) Myo-pericarditis.                       |
|   | (c) Endo-myo-pericarditis.                  |
| 2. PERICARDITIS, with intrinsic complications ...         | (d) Tuberculous deposit in the pericardium. |
|   | (e) Cancerous ditto.                        |
|   | (f) Hæmo-pericardium.                       |
|   | (g) Pneumo-pericardium.                     |
|   | (a) Rheumatism.                             |
|   | (b) Tuberculosis.                           |
|   | (c) Pleuritis.                              |
|   | (d) Pneumonia.                              |
|   | (e) Pleuro-pneumonia.                       |
|   | (f) Renal disease.                          |
|   | (g) Hepatitis.                              |
|   | (h) Scorbutus.                              |
| 3. PERICARDITIS, with extrinsic complications, viz., with | (i) Gout.                                   |
|   | (j) Phlebitis.                              |
|   | (k) Septicæmia.                             |
|   | (l) Typhus fever.                           |
|   | (m) Enteric fever.                          |
|   | (n) Intermittent fever.                     |
|   | (o) Scarlatina.                             |
|   | (p) Small-Pox.                              |
|   | (q) Measles.                                |
|   | (r) Delirium tremens.                       |

The history, diagnosis, and treatment of pericarditis will be most conveniently discussed under the two first mentioned heads; viz., simple pericarditis, and pericarditis with intrinsic complications. The consideration of the extrinsic complications of the disease will furnish matter for a distinct section.

*Simple acute pericarditis*, usually of rheumatic origin, as already shown, and most frequently met with in young subjects,\* is manifested in the great majority of cases, when asso-

\* Doctor Fuller declares, and in this opinion few, if any, will be found to differ from him, that rheumatic pericarditis is more common in the acute than in the chronic form of rheumatism; in its early than in its late stages; in youth than in advanced age; in females than in males; in persons of an irritable, than in those of a phlegmatic temperament; in the weak than in the strong; and in the fibrous than in the synovial form of rheumatism. Non-rheumatic pericarditis is more common in old age, and in men, manifestly because of its frequent association with Bright's disease; and, finally, he adds, rheumatic pericarditis is more severe, the mortality amounting to 1 in 5·8 cases, and seldom latent; whereas that of the non-rheumatic form is often latent.

ciated with rheumatism, within the first few days of the articular engagement. I have, however, known it to occur as late as the twenty-seventh day from the commencement of articular pain and swelling. In several instances the invasion of pericarditis was announced by precordial distress and accelerated breathing, at least twenty-four hours before physical signs of any kind were perceptible. Drs. Stokes and Mayne, as previously stated, have recorded examples of pericarditis in which symptoms sufficiently characteristic to warrant in their judgment a positive diagnosis, preceded all physical signs by periods varying from thirty to forty-eight hours.\*

I am satisfied that, with few exceptions, the rule is as above stated; namely, that the symptoms of pericarditis take precedence of the physical signs; but, I cannot regard them as sufficiently distinctive to warrant a positive diagnosis of pericarditis, pending the development of physical evidence. Indeed, these symptoms, however suggestive of pericarditis, and in association with acute or subacute rheumatism they are in the highest degree suggestive of that affection, may, individually and collectively, be found to depend upon other causes.

The symptoms of simple acute pericarditis, in its first and second stages, are of a comparatively mild character. The grave and urgent symptoms, such as aggravated dyspnoea, failure of the pulse, oppression at the precordium, indescribable anguish and fear of impending death,† belong rather to those cases in which the muscular substance of the heart is involved in the inflammatory process, or the organ is impeded by a copious effusion of serum into the pericardium. There is, however, more or less decided pain in the precordium. The action of the heart is accelerated, the impulse is sharp and abrupt, and the sounds, especially the second, are clear and ringing, with the addition of a characteristic rustle, distinct from friction-sound. The radial pulse is regular,‡ and rarely above 120 in the minute.

\* See page 326.

† Broussais (*Commentaires des Propositions de Pathologie*, 1829, tom. i., p. 397), attributes this symptom to failure of the circulation in the brain.

‡ I cannot subscribe the opinion of Louis, that irregularity of pulse is characteristic of simple pericarditis. I believe that when associated with pericarditis, irregularity of pulse indicates extension of inflammation to the substance of the heart.

Respiration is quickened in proportion to the increased rate of the heart's action; and there is usually harsh, dry, teasing cough, not accounted for by reference to the lungs, and due, probably, to reflex irritation of the larynx and bronchial surface through the coronary plexuses.

Doctor Stokes truly remarks: "If we except the pain which so commonly attends serous inflammations, the remaining symptoms of pericarditis are to be referred less to the pericardium than to the muscular fibre;"\* and Bouillaud is of opinion that rheumatic pericarditis is often painless, or very slightly painful, when not complicated with pleurisy.†

Doctor Mayne has directed special attention to epigastric tenderness as a symptom of pericarditis. It is most acute when pressure is made upwards in the direction of the heart, and was observed in a large proportion of his cases.‡

In reference to this symptom, Dr. Stokes justly remarks that its value as an aid in the diagnosis of pericarditis is much lessened by its presence, in a form scarcely, if at all, distinguishable from the latter, in acute pleuritis of the left side, and in acute gastritis. I do not think there is anything specific in this symptom, which may be noticed not only in the connexion mentioned by Dr. Stokes, but likewise, and more frequently, in cases of hepatic congestion from any cause.

The absence of pain is more characteristic of pericarditis in the complicated than in the simple form, according to Stokes; and when present it has been occasionally observed to differ from that of pleurisy, in not being aggravated by deep inspiration, or by change of posture. I have found this last observation true in a small percentage of cases. It may be readily explained by reference to the anatomical relations of the inflamed surfaces in both cases.

If the attack complicate acute or subacute rheumatism, its general symptoms are in a great degree masked by that disease. Thus, the expression of anguish stamped upon the face, and possibly attributed to cardiac complication, may be not less rationally

\* *Opus citat.*, p. 1.

† *Traité Clinique des Maladies du Cœur*, 1835, p. 454.

‡ *Loco citat.*

accounted for by the presence of articular pain; and, similarly, heat of skin, and accelerated pulse and respiration, by the general febrile action characteristic of rheumatic fever. The peculiar abrupt impulse of the heart is, however, in association with rheumatism, eminently suggestive of incipient cardiac engagement. In the first stage of pericarditis, or that of vascular injection and suspended secretion of the serous surface, the auscultatory signs are negative, with one exception; namely, the existence of a partially suppressed *rustle*, not amounting to *frottement*. This is so characteristic that, in several instances, guided by it mainly, I have been able positively to diagnose the proximate advent of pericarditis.

The period during which this sign may continue I have known to vary from twelve to forty-eight hours; usually it lasts about twenty-four hours before friction-phenomena are declared.

The occurrence of friction-sound, with or without tactile vibration, proclaims the advent of the second stage characterized by exudation of lymph or fibrin.

Doctor Gairdner, however, is of opinion that "the friction-sound in some, even in many cases, may be the indication only of a more partial and evanescent morbid action than any to which the name of acute inflammation could with propriety be applied."\*

I have already discussed at length this phenomenon (p. 289) and shall here only repeat that it is characterized by harshness of quality, prevalence at base, sensible proximity to the ear of the auscultator, liability to intensification by precordial pressure and by full inspiration, and inconstancy as to site, rhythm, and intensity.

The phenomena with which it is especially liable to be confounded are endocardial murmur, and pleuritic friction-sound limited to the antero-inferior portion of the left side of the chest.

For the distinction between endocardial murmurs and the attrition-sounds of pericarditis, Skoda† relies, on the one hand, upon the general effects of valvular disease upon the circulation

\* *Edinburgh Monthly Journal*, 1851.

† *A Treatise on Auscultation and Percussion*; translated by Markham, 1853, p. 225.



and the heart, and, on the other, upon the coexistence of percussion-dulness in pericarditis.

But surely, in the early stages of valvular disease, when alone the question of diagnosis from pericarditis can arise, the derangements of circulation and the alteration in the volume and action of the heart, upon which exclusively he would base a positive diagnosis of valvular lesion and the absence of which he would regard as conclusive against that view, are, in many instances, either entirely absent, or present only in a degree quite insufficient to warrant a positive opinion.

Again, many of those changes, having reference at least to the circulation, are notoriously liable to occur in the course of pericarditis associated with extension of inflammation to the muscular substance of the heart, or with serous effusion into the pericardium.

As to precordial dulness from liquid effusion, as a means of determining an otherwise doubtful murmur to be of pericardial origin, I regard the *absence* of dulness from that cause, where attrition-murmur exists, as the rule, and its presence as the exception.

Skoda denies that the character of the murmur itself is capable of aiding our diagnosis; an opinion in which few physicians will be found to agree with him. He adds: \* "If percussion indicates the presence of effusion in the region of the heart, and if the heart be not thereby displaced, we may conclude that the effusion is in the pericardium; and it then becomes very probable that the murmur also proceeds from the internal surface of the membrane." But if there be no palpable increase in the area of precordial dulness, and that in the majority of cases of pericarditis when friction-sound is audible there is no such increase I positively aver, how is the diagnosis to be made? Are we reduced to probabilities in the determination of this important question of practical medicine? I incline to think few will answer now in the affirmative.

Doctor Hope† relies upon the rougher quality of the pericardial sound as distinguishing it from those due to valvular lesion; and so well pronounced is this difference, that, according to him, where the two kinds of murmur coexist in the same sub-

\* *Opus citat.*, p. 226.

† *Opus citat.*, p. 174.

ject "the one may be heard *through* the other." In such a contingency, however distinctive may be the quality of the murmur in most instances, seeing that it is remarkably prone to variation in this respect, I would rather rely upon other characteristics for its identification; and, pre-eminently, upon its rhythm, site, and liability to change of situation and character within brief periods of time.

Hope also holds that a rough, creaking, or rasping murmur with the second sound must be due to attrition, as a valvular murmur of diastolic rhythm is never of this quality. To this doctrine there are very few exceptions, notwithstanding that Dr. Walshe holds an opposite opinion.\*

Da Costa declares that it is sometimes impossible to distinguish a pericardial from an endocardial sound.† I hold, on the contrary, that it is always possible to make the distinction with confidence, by carefully attending to the characteristics of pericardial *frottement* already indicated. Although in many cases, apparently very difficult, I have found the differential diagnosis in all instances feasible, at least after a second examination.

The coexistence of tactile fremitus with attrition-murmur in pericarditis, though certainly the rule, is not without exception. Enfeeblement of the heart, from any cause, by lessening its contractile energy, may entirely suspend tactile vibration, whilst only modifying, or rendering less loud and harsh, the sound of attrition. Again, fremitus, though present, may not be communicated to the hand, owing to impairment of the vibratile properties of the chest-wall, as, for example, by superficial cedema; and the intervention of emphysematous lung-substance between the pericardium and the wall of the chest, may mask a pericardial *frottement* capable of producing sensible thoracic vibration.

In many cases of valvular murmur exclusively, fremitus is also present. This is notably true of the presystolic murmur of mitral constriction. The distinction, therefore, between pericardial friction-sounds and valvular murmurs, based upon the pre-

\* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 118. In the fourth edition of his work just issued (1873), Dr. Walshe, however, states that aortic regurgitant murmur, the type of diastolic murmurs, is "rarely rough."

† *Medical Diagnosis*, third edition, 1870, p. 357.

sence or absence of *frémissement*, possesses only a limited value ; and, independently of other considerations, excluding certain forms of valvular disease, *frémissement* affords, at least, but very equivocal evidence of pericarditis.\*

The duration of friction-sound may vary from a few hours to three weeks. The period of its continuance is usually very brief where the cause of its suspension is serous effusion into the pericardium. In such cases it will be noticed to cease at the lower part of the precordium in the first instance, and thence to be gradually extinguished in the direction upwards, *pari passu* with the establishment of percussion-dulness.

I cannot concur in opinion with Dr. Gee, to the effect that in cases of serous effusion into the pericardium, consecutive to pericarditis, the liquid accumulation takes place in the first instance at the base of the heart and around the roots of the great vessels.†

In a few instances friction-sound continues to be heard at the extreme upper portion of the precordium, throughout the stage of serous effusion, and whilst the remaining portion yields femoral dulness, and the heart's sounds are everywhere masked. These are cases, however, in which the effusion has fallen short of distension of the pericardium ; and the liquid gravitating to the lower portion of the sac, its superior anterior portion is allowed to come into contact with the roots of the pulmonary artery and

\* Doctor Stokes, however, insists upon a perceptible difference in quality between the *frémissement* of pericarditis and that of valvular lesion ; adding, that the fremitus of valvular murmur is, like the murmur itself, limited to the areas of the respective orifices, whilst that of pericarditis is more diffused, and indifferent to fixed areas. This is a very just distinction so far as it applies ; but circumscribed and persistent pericardial *frémissement* may be limited to the area of one of the orifices, though I admit such is not usually the case. Again, *frémissement* arising from aneurism connected with the intra-pericardial portion of the aorta, may exhibit no relationship to valvular areas, although of limited extent. Doctor King Chambers (*Lectures chiefly Clinical*, fifth edition, 1865), lays down the following rule for the distinction of pericardial friction-sound from valvular murmur. If the ear be withdrawn from the stethoscope while still held in contact with the surface in the site of the sound under investigation, so that the lobe shall barely touch the instrument, the normal cardiac sounds will be heard, *free from murmur* in the case of pericardial friction, but *associated with murmur* if valvular disease be the cause of the phenomenon. I have put this rule to the test, and have found it practically useful.

† *Auscultation and Percussion*, 1870, p. 247.

aorta. If the shoulders be depressed whilst the patient lies upon the back, the situation of friction-phenomena will, in such cases, be reversed.

In a few examples, to be noticed farther on, I have known the friction-sound of pericarditis to continue after the restoration of the patient to apparently perfect health. The rheumatic attack was of a mild character, and no symptoms were referable to the heart; nevertheless a distinct friction-sound, circumscribed within an area of very small extent, was audible at the base of the heart, continued for several weeks, and was unaltered in rhythm or intensity at the time of the patients' discharge.

*Tactile fremitus* is liable to considerable variation in degree of intensity, and seldom amounts to the *frémissement cataire* of Laennec. It is due to vibration of the framework of the chest, consequent upon, and therefore isochronous with, the movements of the heart. It indicates a considerable degree of roughness of the opposed serous surfaces, and hardness of the effused lymph. Dr. Stokes is of opinion that a dry condition of the surface has the effect of intensifying fremitus, if it does not constitute an essential condition of its presence. This phenomenon is never coeval with friction-sound, commencing later, and terminating earlier than the latter.

*Adhesion* of the parietal pericardium to the surface of the heart may follow immediately the dry stage, without the intervention of serous effusion. When it is in process of establishment, the friction-sound is irregular in rhythm, intermittent, and lowered in intensity; it next becomes single and systolic in rhythm; ceases to be heard in one or more situations within the precordium, usually persisting longest at the base of the heart, but occasionally at the apex, and is finally extinguished. The process of adhesion, where serum in notable quantity has not been effused, may commence and be completed within a period of twenty-four hours.

The *positive* diagnosis of cohesion between the parietal and visceral pericardium, from evidence afforded by that condition itself, independently of personal observation of the previous illness, or a reliable history of its progress, is not possible in the present state of the science of physical diagnosis.



Palpitation, supposed to indicate irritative reaction under restraint, was long regarded as a symptom of adhesion of the pericardium to the heart. Morgagni,\* however, after carefully analysing a great number of reported cases of palpitation alleged to arise from this cause, and also several cases of adherent pericardium noted by Valsalva and himself, arrives at the conclusion that palpitation cannot be fairly attributed to adhesion of the pericardium *alone* in the adduced cases; in all of which, other, and frequently many, causes combined to produce it. He observes, moreover, that, with a single exception, in all the cases witnessed by himself in which this was the only tangible morbid condition discovered, palpitation was *not* present. The exception was that of a patient who had suffered from palpitation, and in whom, after death, adhesion of the pericardium to the roots of the great vessels was found. To this latter circumstance, as tending to obstruct the outflow of blood by constricting the vessels, rather than to the general adhesion of the pericardium, he inclines to attribute the palpitation which had been observed during life.

Senac† also doubts that adhesion of the pericardium has any *special* influence in causing palpitation, which he attributes in a general way to various forms of external and internal irritation of the heart, of which adherent and thickened pericardium may be one; especially if, at the same time, it make pressure on the roots of the great arteries.

Corvisart‡ regards, as symptoms of adherent pericardium, frequent flushings, and a sensation of dragging in the region of the heart, due to downward traction of the pericardium and heart by the diaphragm during inspiration, and upward traction of the diaphragm by the heart in systole. He mentions three kinds of adhesion; namely, 1, by exuded lymph; 2, by direct union without a uniting medium, which he regards as the usual form in gout and rheumatism; and 3, long filamentous bands.

Skoda, however, holds,§ but not very strongly, that the diag-

\* *The Seats and Causes of Disease*, translated by Alexander, book ii., letter xxiii.

† *De la Structure du Cœur*, l. iv., ch. xi.

‡ *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels*, translated by Hebb, p. 32.

§ *Opus citat.*, p. 327, note.

nosis is practicable ; he bases it mainly upon suppression of apex-pulsation, and an alleged upward and inward displacement, with fixation, of the apex of the heart. He mentions as a further sign of adhesion, systolic retraction of the intercostal space at a point corresponding to the apex, and to a slight extent of one or two spaces next above ; but this implies adhesion of the pericardium to the costal pleura.

I cannot regard these signs, taken absolutely, as of much value. Displacement of the apex upwards and inwards may be the result of partial retraction of the left lung, already adherent to the cone of the pleura and to the external surface of the pericardium ; whilst suppression of the apex-beat may be due to fatty disease of the heart, or enfeeblement of the organ from other causes, such as typhus, or septic blood-poisoning. But if the other causes of upward displacement of the heart be excluded, and especially if pericarditis be known to have preceded, a fixed position of the apex-point at or near the nipple may be regarded as eminently suggestive of adhesion.

Laennec alludes\* to the sign mentioned by Sanders ; namely, retraction or pitting of the epigastrium below the left costal cartilages, followed by elevation with pulsation of the same point. He declares, however, that although he had repeatedly looked for it where adhesion of the pericardium actually existed, he had never observed it.

This is likewise my own experience. I have repeatedly observed pericarditis pass through its entire course, from the pre-friction or dry stage, to final suppression of friction-sound, and I have not even once witnessed the sign mentioned by Sanders. I have frequently observed epigastric pulsation of such a character as might be readily mistaken for the phenomenon in question ; but, I feel bound to add that, I have observed this most frequently in hypertrophy with dilatation, where no adhesion of the pericardium existed.

Laennec does not consider the diagnosis feasible. Such is likewise the opinion of Bertin and Bouillaud.

Hope, while asserting that the diagnosis of adherent pericardium cannot be made with positive certainty, considered that

\* *Mediate Auscultation*, Forbes' translation, p. 666.

strong probability of its presence may be assumed, if, with hypertrophy of the heart, there be abnormal prominence of the precordium, without downward displacement of the organ; and, at the same time, a jogging or tumbling movement of a double character, synchronous with the sounds of the heart. This latter he regards as being, in a diagnostic sense, the more valuable of the two signs, and as warranting a very strong presumption of cohesion of the pericardial surfaces.\*

I cannot attach the importance to this sign which Dr. Hope has done, as evidence of adhesion of the pericardium. I have repeatedly found it present where no adhesion existed, as proved by examination of the body after death; and I have rarely missed it where considerable hypertrophy with dilatation of the left ventricle had taken place (case of John Hogan). In the latter case, unless the hypertrophy be associated with fatty metamorphosis, there is invariably a second or *diastolic* impulse present (case of William Hogan). This I consider due to the energetic character of ventricular expansion at the commencement of diastole, combined with the extensive range of its forward movement; and I incline to regard it as identical with the "jogging movement" of Hope.

Doctor Stokes has likewise failed to identify this peculiar movement of the heart, in connexion with adhesion of that organ to the parietal pericardium.

The diagnosis is not attended with equal difficulty when, at the same time, adhesion of the pericardium to the chest-wall and to the diaphragm exists. Under these circumstances systolic dimpling of the cutaneous surface at the point of apex-pulsation, and, generally, to the extent of endo- and exo-pericardial adhesion, also coinciding, possibly, with pitting at the epigastrium and retraction of the lower end of the sternum, may be observed.

According to Friedreich,† diastolic collapse of the jugular veins will be found in many cases to alternate with these phenomena; owing, as he surmises, to elongation of the superior cava, and consequent suction of the jugulars during the descent of the

\* *Opus citat.*, p. 194.

† Niemeyer's *Text-Book of Practical Medicine*, 1869, vol. i., p. 391.

diaphragm after systole. If adhesion of the pericardium to the adjacent portions of the anterior wall of the chest exist, the edges of the lungs will be precluded from advancing, as in the normal state, during inspiration; and the area of superficial precordial dulness will be found, therefore, to have undergone no diminution at the acme of full inspiration.

Laennec mentions\* dulling of the second sound as evidence of adhesion of the anterior surface of the heart to the pericardium. But this is certainly an error; and it seems an inference from his theory of the causation of the second sound, namely, contraction of the auricles, rather than the result of clinical observation.

I have frequently observed resolution of the first sound to follow pericarditis, when the previous course of the disease forbade all doubt as to the actual existence of adhesion of the pericardium (case of M. Nolan); and I incline to account for this anomaly by reference to the impediment which extensive adhesion must present to the movement of systolic ascent of the apex. But, whatever value the explanation may possess, I am satisfied as to the fact, that recent adhesion of the pericardium to the heart is attended with resolution or reduplication of the first sound, which, in such cases, is likewise somewhat less clear in tone.

Walshe mentions† upward extension of the area of precordial dulness, even to the lower edge of the second costal cartilage, consecutive to pericarditis, as a sign of adhesion.

I have not met with an example of this kind either during or consecutive to pericarditis, where indubitable evidence of liquid effusion into the pericardium did not also exist. I can, however, with Dr. Walshe, conceive displacement of the heart upwards, but in a minor degree, by copious effusion; and its being permanently fixed above its normal level by subsequent adhesion to the pericardium.

I consider, then, that whilst simple cohesion of the parietal and visceral surfaces of the pericardium cannot be diagnosed positively, a very strong presumption of the existence of this

\* *Mediate Auscultation*, Forbes' edition, p. 665.

† *Opus citat.*, p. 47.



condition would be warranted, were the apex of the heart permanently displaced upwards, the first sound resolved and the impulse-element dulled, and a clear history of antecedent pericarditis obtained. If, with or without such history, systolic pitting of an intercostal space at the point of apex-pulsation, and of the epigastrium, with absence of variation in the relative extent of precordial dulness and vocal vibration during full inspiration existed, I should have no hesitation in concluding that both pericardial and pleuro-pericardial adhesion had taken place. Exaggerated systolic depression of the epigastrium, and of the lower end of the sternum, would indicate the twofold condition of general agglutination of the pericardium, and hypertrophy of the heart.

The effect of adhesion of the pericardium upon the heart, and its influence upon the duration of life, were regarded by the older writers as of the most unfavourable character. This was, no doubt, due to their failure to recognise, or at least to appreciate in its full significance, the less striking lesion of the valves; which, coeval with adhesion of the pericardium to the heart, and constituting with it the compound result of long antecedent endo-pericarditis, was either entirely passed over, or received only cursory notice in the examination of the heart after death. The more obvious lesion of the pericardium was promptly recognised, and, as affording an apparently simple and ready explanation of the increased volume of the heart, was associated, in the crude pathology of that time, with the latter change, as cause with effect.

Morgagni held that cohesion of the opposed surfaces of the pericardium was a cause of dropsical effusion.\* Baillie was of opinion that close and general adhesion gave rise to oppression and difficult breathing; whilst Corvisart thought that it must be ultimately fatal by derangement of the function of the heart.†

Bouillaud considered hypertrophy of the heart and œdema to be necessary consequences of simple adhesion of the pericardium, whilst atrophy was a necessary result of compression of the

\* *De Sedibus et Causis Morborum.*

† *On Diseases of the Heart*, translated by Hebb.

heart by thick layers of false membrane.\* Cheevers† and Barlow‡ declare that atrophy of the heart is the usual and normal result of obliteration of the pericardial sac by adhesion; the former adding, that when dyspnœa and dropsy occur in connexion with it, they are due to consecutive contraction of the arterial orifices, and venous engorgement. Barlow admits that engorgement of the right side of the heart, of the veins, and general dropsy, may be the consequences of adhesion of the pericardium; but he traces the connexion between them through impairment of respiration, which he attributes to restricted movement of the ribs and diaphragm.

Complete obliteration of the sac of the pericardium by adhesion, leads inevitably to hypertrophy with dilatation of the heart, according to Hope.§ He regards hypertrophy as the result of increased nutrition and muscular development of the heart, consequent upon the increased efforts in the performance of its function, imposed upon that organ by adhesion of its surfaces. The difficulty under which the heart is now required to “function,” consisting in the impediment to free movement entailed upon it by adhesion of the pericardium to its entire surface, leads, as in the case of all muscles similarly circumstanced, to hypertrophy of its substance. Dilatation, on the other hand, is due to a state of persistent engorgement of the cavities of the heart, and excentric yielding of its walls, partly the result of antecedent inflammation and softening of its substance, but in a still greater degree, of the centrifugal traction to which it is subjected by universal adhesion of its surface, combined with a process of gradual shortening in the elements of the connecting medium. Limited adhesion, engaging only a small portion of the surface of the heart, may, in the opinion of Dr. Stokes, be the cause of localized dilatation of one of the cavities of the heart, through the centrifugal traction exercised by it.||

Beau upholds the same view. Dr. Stokes observes,¶ “With-

\* *Traité Clinique des Maladies du Cœur*, observation iv.

† *Guy's Hospital Reports*, vol. ix.

‡ *Medical Gazette*, 1847.

§ *Opus citat.*, p. 192.

|| *Opus citat.*, p. 13.

¶ *Opus citat.*, pp. 11 and 12.

out denying that a general adhesion may induce hypertrophy and dilatation, experience leads me to doubt that such an effect necessarily, or even commonly, follows the condition indicated. I have often found the heart in a perfectly natural condition, with the exception of an obliterated pericardium." "It has been stated to me by Professor Smith, that he has found general adhesion of the pericardium coinciding with atrophy, or with hypertrophy of the heart, in a nearly equal frequency. In some of the cases of atrophy, the change was simple, consisting essentially in a diminished volume, with perhaps a paler colour of the heart, while in others a true fatty degeneration had commenced. In another series, the heart showed the fatty degeneration invading, more or less completely, the entire of the cardiac walls. And it is a remarkable fact, recorded by the same observer, that he has always found ossification of the pericardium, which we may hold as the extreme of the obliterating process, attended with atrophy of the heart."

Doctors Barlow,\* Cheevers,† and Gairdner,‡ have arrived at conclusions on this subject, not materially differing from that expressed in the foregoing extract, and tending to show that the doctrine, previously stated, as propounded by Hope, is by much too general.

Doctor Gairdner justly criticizes Hope's conclusions, which he shows to have been drawn from erroneous premises; for, in the five cases adduced by Hope as examples of hypertrophy of the heart *consequent upon* adhesion of the pericardium, there were other and less questionable causes of that morbid change in operation; viz., valvular disease in various forms.

It is but right, however, that whilst agreeing in opinion with Dr. Gairdner as to the insufficiency of the evidence upon which Hope's doctrine rests, and regarding that doctrine as essentially erroneous, I should add, that the cases adduced by Dr. Gairdner,§ in justification of his dissent, are to me by no means conclusive. Of his first series of ten cases, three must be omitted

\* *Medical Gazette*, 1847.

† *Guy's Hospital Reports*, vol. ix.

‡ *Edinburgh Monthly Journal of Medical Science*, February, 1851.

§ *Loco citat.*

for want of reference to the actual state of the heart; whilst the remainder, which are examples of adhesion with atrophy, possess not much value, because they are all examples of wasting chronic disease, viz., cancer, phthisis, diabetes mellitus, granular kidney, and cirrhosis of the liver.

In series 2, consisting of two cases, illustrative of adhesion of the pericardium with cardiac hypertrophy, the latter condition was accounted for by the coexistence of valvular disease.

Series 3 (three cases) is composed of examples of adhesion with hypertrophy, and unaccompanied by valvular lesion. Of these, No. 1 is not admissible, owing to an imperfect report of essential details. No. 2 is an example of Bright's disease ("urine albuminous, specific gravity 1.009") with hypertrophy of the heart, and partial adhesion of the pericardium; and in No. 3, in which there were hypertrophy and dilatation, with partial adhesion, a satisfactory examination of the patient had not been made before death, nor was the state of the kidneys mentioned in the report of the autopsy.

Of Dr. Gairdner's thirteen cases of uncomplicated adhesion of the pericardium, there were at least nine in which "not the slightest hypertrophy was present, and in two or three the heart was remarkably small." He concludes that free movements of the heart within the pericardium, are necessary only in emergencies demanding more vigorous action of the central organ of the circulation, such as severe bodily exertion, or mental emotion; and he would have it inferred that it is only when such causes are superadded to adhesion, that hypertrophy of the heart need be apprehended. But, surely, the causes above mentioned, are of themselves capable, independently of adhesion, of giving rise to hypertrophy. Irrespectively, therefore, of the exception which may be taken to the doctrine promulgated, the conclusion would seem unwarranted by the premises. Nor can I acquiesce in the opinion expressed by the same eminent writer, to the effect, that partial adhesion at the apex may be as effective in producing hypertrophy of the heart, as complete and general adhesion.

With the view of contributing in some degree towards the settlement of the much debated question, as to the effect of ad-



hesion of the pericardium upon the nutrition and volume of the heart, I have collected the following statistics, bearing on the subject, from sources whence information of a reliable character, or least open to suspicion, may be reasonably sought. These statistics I have reduced to tabular form, under separate heads, indicating: 1, the rheumatic or non-rheumatic origin of the pericarditis; 2, whether it was accompanied or not by valvular disease; 3, by liquid effusion; and, 4, followed or not by hypertrophy.

The tables are three in number. The first contains, in epitome, the examples of pericarditis published in the Reports of the London Pathological Society; the second, those given in the Reports of the Pathological Society of Dublin; and the third contains a summary of the cases which have come under my personal notice.

PERICARDITIS : TABLE I.

*London Pathological Society.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(1) Prescott Hewett, vol. i. Dec. 6, 1847	—	1	—	—	Hypertrophy and dilatation of right ventricle; left ventricle small and not thickened.	General adhesion of pericardium.
(2) Dr. Williams, vol. i. April 17, 1848	—	1	—	1	Left ventricle and right ventricle dilated	Partial adhesion of pericardium.
(3) Dr. Parkes, vol. ii. Feb. 19, 1849	—	1	—	—	—	Wound of pericardium, from œsophagus, by swallowing a sword; pneumo-pericardium; general exudation of lymph.
(4) Dr. Bennet Jones, vol. iv. Dec. 7, 1852	—	1	—	—	Of left ventricle	Originating in two circumscribed abscesses between liver and diaphragm, which found entrance into pericardium; extensive adhesion.
(5) Dr. R. R. Robinson, vol. iv. Dec. 7, 1852	—	1	Thickening of aortic, & atheroma of the aorta.	1	1	A man, aged forty-three; intemperate; fall from ladder. <i>Post mortem</i> : congenital opening in pericardium, through which left auricular appendix protruded; fatty heart; calcareous coronary arteries; cirrhosis of liver; Bright's disease; partial adhesion of pericardium by bands of false membrane.

(6) Dr. Bristowe, vol. v. March 21, 1854	—	—	1	—	Double pleuropneumonia; exudation of thick layer of lymph; palor and fatty degeneration of outer layer of ventricles, which was in contact with false membrane.
(7) Dr. Habershon, vol. vi. Dec. 5, 1854	1	—	—	And dilatation of left ventricle; dilatation of right ventricle	General adhesion of pericardium; hydatids encroaching upon right auricle and ventricle.
(8) Dr. Cholmeley, vol. vi May 15, 1855	—	1	—	Contract. of mitral orifice, cohesion of valve, and small nodular growths upon it	Pericardium almost universally adherent; heart enlarged and fatty; large aneurismal pouch at apex of left ventricle, walls of which were composed of endo- and exocardium only; an-gina pectoris and calcified coronary arteries; fatty degeneration of outer and inner layers of muscle.
(9) Dr. Quain, vol. vii. Nov. 6, 1855	—	1	—	—	Pericardium partially adherent and calcified; calcareous deposit in aorta and in mitral valve.
(10) Dr. G. Hewitt, vol. viii. Nov. 18, 1856	1	—	1	Mitral valve warty and incompetent	Much effused lymph, but no adhesion.
(11) Dr. Hawkesley, vol. viii. May 19, 1857	—	1	3 oz. of pus	—	Remittent fever, and eruption on skin; thick and vascular layer of lymph on visceral pericardium only.
(12) Dr. Oubr�, for Dr. Boyd, vol. ix. Dec. 15, 1858	—	—	—	—	A lunatic, aged sixty-three; no reliable history; adhesion of pericardium, and small abscess at apex.

PERICARDITIS : TABLE I.—*Continued.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(13) Dr. Markham, vol. ix. April 6, 1858	1	—	Valves generally slightly thickened, and competent	—	—	Graves' disease, aggravated; enlarged thymus, pressing on pulmonary artery and causing systolic murmur; patch of fibrinous transformation in wall of left ventricle, and a similar change, in a greater degree, of left papillary muscles.
(14) Dr. John Ogle, vol. ix. April 6, 1858	—	1	—	—	1	A large cyst beneath visceral pericardium on posterior surface of right ventricle, containing altered blood and laminated clot; judged to be of long standing, and due to rupture of branch of calcareous coronary artery; general adhesion of pericardium with calcareous deposits in false membrane; texture of heart soft.
(15) Dr. Wilks, vol. xi. April 7, 1860	—	1	—	1	—	A boy of fourteen; had scarlatina six months before; had peritonitis and ascites at period of death; pericardium distended with liquid, nearly filled left pleural cavity, displacing lung backwards and inwards, and concealing it entirely, save apex, and contained a pint of clear serum; parietal pericardium was much thickened by deposit of several layers of false membrane, the inner of which was soft; heart similarly covered, the outer layer being soft and the subjacent layers firm; the deepest tightly embracing the heart, and adhering to the visceral pericardium, which was thickened, and sending fibres into substance of heart; heart small for size and age of patient, and



<p>(16) Dr. Bristowe, vol. xi. Dec. 4, 1860</p>	—	1	—	—	1	<p>appeared as if it had been strangled by the firm coating of lymph upon it; valves and endocardium, generally, quite healthy.</p>
<p>(17) Dr. Gibb, for Dr. David, vol. xiii. Oct. 15, 1861</p>	1	—	General endo- carditis	1	1	<p>A boy of thirteen; tubercular pericarditis, ascites, and pleuritic effusion; tubercular deposit in anterior mediastinum; universal adhesion of pericardium; the adhesions thick and fibroid, and in them much tubercular matter, in form of cheesy masses and laminae of great extent; walls of left ventricle thin, and cavity dilated; right ventricle very thick and dilated; auricles dilated.</p>
<p>(18) Dr. Peacock, vol. xiv. April 7, 1863</p>	—	1	No mention	—	No mention	<p>A coloured man, aged forty-two; died in St. Patrick's Hospital, Montreal; acute rheumatism of ten days' duration, with pleuritis and pericarditis; pericardium contained eight ozs. of turbid serum, and both its surfaces were covered with thick dense flakes of fibrin "like tripe"; there was no adhesion. Intense endocarditis on both sides; heart weighed forty-six and a-half ounces; it was fifteen and a-quarter inches in circumference, and six inches in length.</p>
<p>18</p>	4	12	5	5	11	<p>A man, aged thirty-one; carcinoma of lung, intestine, kidneys, and supra-renal capsules; pericarditis with nodules of carcinoma in false membrane. No notice of size of heart, or state of valves, and no information given as to whether there was adhesion of pericardium, or effusion into its cavity.</p>

PERICARDITIS : TABLE II.

*Dublin Pathological Society.*

Cases.	Rheumatic.	Non-rheumatic.	Valvo-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(1) Dr. Law, vol. i. April 3, 1841.	1	—	Aortic and mitral	—	1	Patient aged twenty-two; had repeated attacks of rheumatism; heart weighed thirty-six ounces; complete adhesion of pericardium.
(2) Mr. O'Ferrall, vol. i. April 2, 1842.	1	—	No mention	—	1	A boy of eleven; had scarlatina six months, and rheumatic fever, two months before death; adhesion of pericardium by bands of lymph.
(3) Dr. Corrigan, vol. i. Dec. 3, 1842	—	1	Mitral (?)	1	—	A boy of eighteen; pericard. so much expanded by serous effusion as to reach first rib, and coated internally with rough papillated lymph; slight thickening of mitral valve; no adhesion.
(4) Dr. Corrigan, vol. i. Jan. 13. 1844	—	1	No mention	1	No mention	A railway labourer; pericarditis of four months' duration; three pints of dark-coloured serum in pericard. and some adhesion at base; heart rough by effused lymph; "pine-apple heart."
(5) Dr. Lees, vol. i. Jan. 11, 1845	—	1	No mention	1	No mention	A woman of sixty-eight; left circumscribed pleuritis with pericarditis; hemorrhagic effusion into pericard. which was reticulated; no adhesion.
(6) Dr. Corrigan, vol. ii. Dec. 7, 1850	—	1	No mention	1	No mention	A girl of seventeen; overworked; died of dysentery; pericarditis diagnosed by <i>frottment</i>

audible when she lay on her back, but inaudible when she sat up and leaned forward; a few flakes of unorganized lymph were found, and a few patches, organized, on the outersurface of the right ventricle. Eight ounces of liquid in pericardial sac. The history showed that the lymph had remained unorganized for a period of six weeks; no adhesion.

A man of sixty; pleuro-pneumonia; heart enlarged and fatty; slight atheroma of aorta; calcareous plate, two inches diameter, in false membrane on right ventricle; nearly complete adhesion of pericardium.

A boy of twelve; had acute rheumatism three years before; chorea, with mitral reflux and hypertrophy, a year later; at end of another year dyspnoea, pericardial *frottement*, and friction-sound. Heart covered, except at apex, by several layers of false membrane, in some places three-quarters of an inch thick; adhesion by bands of false membrane at base.

A boy of thirteen; acute rheumatic endocarditis at eleven; a year and nine months later another similar attack, and five months subsequently died in syncope; universal adhesion of pericardium.

An infant of five months; pericarditis after measles; pericardium very vascular, and filled with pus; no adhesion.

(7)  
Dr. Banks, vol. ii.  
Jan. i. 1848

(8)  
Dr. McDowel, vol. iii.  
April 16, 1853

(9)  
Dr. Mayne, vol. iii.  
March 31, 1855

(10)  
Dr. Mayne, vol. iii.  
Same date

PERICARDITIS: TABLE II.—Continued.

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(11) Dr. M'Dowel, vol. iii. Dec. 5, 1856	1	—	Slight thickening of aortic and mitral	1	—	A child of eleven years; death on the ninth day; a few ounces of serum in pericardium; surfaces vascular; some false membrane; no adhesion.
(12) Dr. Henry Kennedy, vol. iii. Jan. 8, 1859	No mention	No mention	—	—	1	There was hypertrophy, with dilatation, and fatty disease of substance of heart; nearly complete adhesion of pericardium.
(13) Dr. Hudson, vol. iii. April 20, 1861	No mention	No mention	No mention	1	No mention	Pericarditis, with inflammation; softening of the heart; friction-sound ceased three or four days before death. <i>Post mortem</i> : serum with coating of lymph; softening of heart, and infiltration of its substance; "grumous fluid." Dr. Hudson remarks that the signs of extension of inflammation to the heart were, increased precordial pain and anxiety, tendency to syncope, failure of radial pulse, irregularity of heart's impulse, and cessation of attrition-sound without increase of precordial dulness.
(14) Dr. Banks, vol. iii. Dec. 6, 1862	No mention	No mention	Aortic and mitral inadequacy from dilatation	—	1	A man, aged eighteen years; hypertrophy to thirty-eight ounces with dilatation; universal adhesion of pericardium.



(15) Dr. Henry Kennedy, April 16, 1864	No mention	No mention	No mention	—	No mention	Acute pericarditis, without the usual accompaniment of <i>frottement</i> , although pericardium was covered with reticulated false membrane. Dr. Kennedy inquires whether this may not have been due to rapid action of heart. No adhesion.
(16) Dr. Gordon, vol. iii. March 21, 1868	I	—	—	I	With dilatation	Secondary pericarditis, with exo-pericarditis; exo-pericardium adherent to chest-wall; hence says Dr. Gordon, the great dilatation with hypertrophy; whereas, in ordinary adhesion with compression of the heart, atrophy is the rule; adhesion.
(17) Dr. Lyons, vol. iii. March 30, 1867	—	I	No mention	I	No mention	Acute pericarditis in the fourth week of typhus, and fatal on the twenty-seventh day; serum and lymph were found in pericardium; no adhesion. (?)
(18) Dr. Hayden, vol. iii. May 2, 1868	—	I	—	—	—	A man, aged twenty-two; hepatic disease and general dropsy; cartilaginous and calcareous transformation of pericardium; atrophy and dilatation of heart; general adhesion.
(19) Dr. Jennings, vol. iv, part. i. Jan. 9, 1869	No mention	No mention	—	—	I (?)	A man, aged forty-two; sudden death; tuberculosis of lungs and pleural effusion; external adhesion of pericardium; left ventricle fatty; aorta highly atheromatous; complete adhesion of pericardium by means of false membrane half an inch thick.

PERICARDITIS: TABLE II.—*Continued.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(20) Dr. Eames, vol. iv. part i. Jan. 16, 1869	1	—	—	1	1	A child at nurse, neglected; pericardium extended to right nipple, and upwards to second rib on left side; it had pushed up lung above level of clavicle; it contained twelve ounces of amber-coloured serum, and was covered with false membrane; purulent deposits in various parts; no adhesion.
(21) Dr. T. E. Little, vol. iv. part i. Jan. 16, 1869	—	1	—	1	—	A man, aged thirty-one, with curvature of spine, an inmate of the Sligo Lunatic Asylum; signs of pericarditis had continued three months; pericardium twenty-four inches in circumference; vascular externally, thickened, and coated internally with lymph, and containing sixty-five ounces of dark-coloured serum; mediastinal glands large and tubercular; left pleural adhesion; no pericardial adhesion.
(22) Dr. Stokes, vol. iv. part i. Jan. 30, 1869	1	—	Double aortic	—	1	A man twenty-five years of age; long subject to yearly attacks of rheumatic arthritis; a ploughman by occupation; double murmur at aortic orifice; throbbing of carotids, and loud pericardial <i>frottement</i> ; both aortic murmurs disappeared before death; whereas, according to Dr. Stokes, this is rarely the case with regard to the aortic diastolic murmur; pericardium was found greatly thickened and covered with

<p>(23) Dr. MacSwiney, vol. iv. part i. Jan. 29, 1870</p>	<p>1</p>	<p>—</p>	<p>Recent de- posit on aortic and mitral</p>	<p>—</p>	<p>No mention</p>	<p>lymph in great quantity, disposed evenly, and in rugged masses; one segment of the aortic valve was calcified and incompetent; general hypertrophy; heart, with pericardium, weighed four pounds and two ounces; the left ventricle was one and a-half inch thick; partial adhesion of pericardium. Dr. Stokes is of opinion that the great and general hypertrophy in this case was due to the laborious occupation of the poor man, and which he himself thought had agreed with him.</p> <p>A man who had had rheumatism four years previously, was attacked with pleuro-pneumonia and pericarditis; pericardium was found thickened, and both its surfaces coated with rough lymph; foetid gas escaped on opening the pericardium, which, having been distended, was supposed to contain liquid; partial adhesion of pericardium.</p>
<p>23</p>	<p>9</p>	<p>9</p>	<p>8</p>	<p>10</p>	<p>11</p>	<p>23</p>

PERICARDITIS : TABLE III.

*The Author's Cases.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(1) J. Doyle	1	—	—	1	—	Pericarditis twelve days after rheumatic invasion ; no head symptoms ; recovery.
(2) J. Doran	1	—	Regurgitant, mitral, double aortic	—	1	Rheumatism sixteen years ago, and pericarditis with return of articular pains ; no head symptoms ; recovery.
(3) L. O'Brien	1	—	Mitral obstruction and reflux	1	1	Pericarditis seventeen days after articular pains ; no head symptoms ; recovery.
(4) Pat. Macdonnell	—	1	Double mitral, and aortic, and tricuspid	1	1	Had rheumatism nine years before, and again several times, but not recently ; no head symptoms.
(5) Pat. Costello	—	1	Mitral obstruction	—	1	Long subject to rheumatic gout, but pericarditis consecutive to organic disease ; no head symptoms ; death.
(6) Margaret Bligh	—	1	—	1	1	Palpitation one year before, after retrocession of cutaneous eruption ; no head symptoms ; recovery.



(7) Mary Nolan	1	—	Temporary mitral in- adequacy	1	—	Pericarditis two days after articular pains ; murmur had entirely ceased when discharged ; slight nocturnal delirium.
(8) John Smith	—	1	—	1	1	Pulmonary emphysema, and hypertrophy of right ventricle ; head symptoms due to this ; death ; no <i>post mortem</i> .
(9) Anne Lalor	1	—	Mitral re- flux	1	1	Had had rheumatism ; hypertrophy judged by heaving impulse ; no <i>post mortem</i> ; murmur de- veloped in last illness ; no head symptoms.
(10) Kate Kavanagh	1	—	—	1	—	Recent case.
(11) James Walsh	1	—	Double aortic and single mitral	1	1	Pericarditis, not preceded by articular swelling, four days after exposure ; valve-disease of long standing, as judged from hypertrophy of heart.
(12) Pat. Moore	1	—	—	—	—	Recent case.
(13) James Farrell	—	1	—	1	1	Chronic phthisis, pericarditis and pleuritis ; no adhesion.
13	8	5	7	10	9	13

Of the preceding cases, in number 54,

Valvular disease did not exist in 23.

Of this latter number hypertrophy was present in 14 :

General adhesion ... ..	8
Partial adhesion ... ..	2
Calcification of false membrane ... ..	4

Hypertrophy did not exist in 7.

Atrophy existed in 2 :

Strangulation by thick layers of false membrane ... ..	1
Cartilaginous and calcareous transformation of false membrane ...	1

Of the cases in which hypertrophy did not exist, there was no adhesion in 5

„ „ „ „ adhesion in 2

„ „ „ „ recovery in ... 2

Of the total of cases, in number 54 :

Hypertrophy was present, associated with adhesion of the pericardium, and unconnected with any other assignable cause of its existence, such as chronic valvular or renal disease, or arterial atheroma, in 7

Valvular disease existed in 20 :

Thickening of valves generally ... ..	2
Mitral, aortic, and tricuspid obstruction and reflux ... ..	1
Aortic obstruction ... ..	1
Aortic obstruction and reflux ... ..	1
Mitral obstruction ... ..	2
Mitral reflux ... ..	6
Mitral obstruction and reflux ... ..	1
Mitral reflux, and aortic obstruction and reflux ... ..	6
No mention is made of the condition of the valves in ... ..	9
There was temporary mitral regurgitation in ... ..	1
There is doubt as to the condition of the valves in ... ..	1
Pericarditis was associated with rheumatism in ... ..	21
„ „ not associated with rheumatism in ... ..	26
Hydro-pericardium existed in ... ..	25
Hypertrophy in ... ..	31

Hypertrophy did not exist in 9 :

No adhesion of the pericardium ... ..	5
Adhesion of the pericardium ... ..	2
Recent cases ; recovered ... ..	2

From the preceding data, it appears that out of a total of fifty-four cases of pericarditis, collected from the three sources indicated, only seven can be adduced in support of the doctrine that simple adhesion of the pericardium is competent to give rise to hypertrophy of the heart ; but, even these are not re-

ported in such a manner as to inspire complete confidence. In several of them the urine was not tested, nor was disease of the kidneys excluded by necroscopic examination. I therefore feel warranted in concluding, as I had been for a long time led to infer from personal observation, that, contrary to the teaching of Hope, when hypertrophy of the heart is met with in association with adherent pericardium, the connexion is, in the majority of cases, purely accidental. Some other less questionable cause of hypertrophy is present, such as valve-lesion, chronic renal disease, or atheroma of the aortic arch. Dr. Barclay is of the same opinion, regarding aortic atheroma and renal disease as the cause of simple hypertrophy of the heart in the majority of cases.\* But, even with this deduction, examples of hypertrophy are to be met with, and a few such are included in the preceding list, in which increase in the weight and volume of the heart would seem to depend upon adhesion of the pericardium exclusively. How are such cases to be explained? I believe that the greater number, if not the entire, of such cases, resolve themselves into examples of pericarditis occurring in infancy or childhood, in a robust constitution, and where no mechanical impediment to the growth of the heart existed. Under these circumstances, the persistent stimulus to nutrition, from inflammatory irritation of its surface in the first instance, and, subsequently, from the mechanical irritation of inflammatory products to which the heart is subjected, must issue in excessive development of its substance, provided only the conditions for a proportionate increase in the supply of pabulum, and the capacity for its appropriation by the tissue of the heart, be likewise present.†

\* *Medico-Chirurgical Transactions*, vol. xxxv.

† Dr. Moxon (Lectures on Analytical Pathology, lecture ix., *Medical Times and Gazette*, November 26th, 1870), is of opinion that "under irritation the tubular elements (nerve, muscle, capillary vessel) never multiply, but waste away, or rapidly break down." But, surely, under irritation physiological activity is quickened, and exaggerated structural development is thereby induced.

Dr. Todd (*Clinical Lectures by Beale*, 1861, lecture xvi. p. 482) holds that hypertrophy and dilatation of the heart are usual pathological sequences of adhesion of the pericardium. I cannot agree in this opinion. For the discussion of the subject generally, the reader is referred to the chapter now under consideration.

If the coronary arteries be free from obstruction, and the heart be not constricted by false membrane of a thickness and power of resistance sufficient to prevent its growth, then simple pericarditis, occurring within the first or second period of life, when nutrition is active, growth rapidly progressive, and buoyancy of spirits inductive of vigorous movements of the body, will inevitably lead to hypertrophy of the heart. I believe that these arguments apply, with scarcely less force, to the period of adolescence, provided only the constitution be vigorous.

If, on the other hand, even in the early periods of the life of healthy subjects, pericarditis give rise to constriction of the nutrient vessels of the heart, of its substance, or of both, arrest of growth, or atrophy, will be the result, irrespectively of the character of the constricting medium.

Bouillaud is of opinion\* that adhesion, as such, does not interfere with the play or function of the heart.

Doctor R. W. Smith,† holds that calcification of the pericardium is more often associated with atrophy than with hypertrophy of the heart. He published the case of a man aged thirty-nine years, in whom the ventricles were encircled by an osseous belt, one inch broad, imbedded in their substance, the auricles were hypertrophied, but the ventricles had undergone no particular change.‡ In a case of ossified pericardium, Louis found the volume of the heart a little reduced.§

Abercrombie gives the following particulars of a case of pericarditis with adhesion and ossification of the false membrane. A man, aged forty-two, after exposure to cold and fatigue, exhibited symptoms indicative of acute intrathoracic inflammation, from which he recovered. He subsequently suffered from dyspnoea, palpitation, and general dropsy, and four years after the date of first illness he died. The autopsy revealed extensive adhesion of the pericardium to the surrounding structures, also general adhesion of the pericardium to the heart, and, in the uniting medium, osseous plates; "the heart

\* *Traité Clinique des Maladies du Cœur*, 1835, p. 454.

† As quoted by Dr. Stokes.

‡ *Dublin Journal of Medical Science*, vol. ix. p. 411, July 1st, 1836.

§ *Memoires ou Recherches Anatomico-Pathologiques*, 1826, p. 298.



was not at all enlarged, or in other respects diseased." He adds the opinion that dilatation supervenes on inflammation of the heart, and is usually accompanied by adhesion of the pericardium.\*

Doctor W. Budd declares† he has seen many cases of adhesion, often general, without organic alteration of the heart, or impairment of its function.

Bouillaud expresses an opinion to the effect that a heart may be reduced to half of its volume, or even less, by the compression of thick layers of false membrane.‡ Walshe is of opinion that atrophy of the heart may result from thick layers of false membrane firmly embracing the heart, and pressing on the coronary arteries so as to interfere with the circulation through them.§ Finally, Dr. Barclay declares that adhesions of the pericardium are more frequently associated with dilatation than with hypertrophy.||

From the preceding evidence, the conclusion seems inevitable, that the doctrine of hypertrophy from simple adhesion can no longer be maintained.

Abercrombie, discussing the effects upon the heart of adhesion of the pericardium, says, "In the cases which have gone on for a considerable time, the heart is generally enlarged; this enlargement seems to be, in many cases, confined to the left ventricle, and to consist of an enlargement of the cavity, without thickening of the parietes." Yet the case of enlargement which he gives, presented valve lesion also, as shown by dissection. He adds, "but enlargement may exist without adhesion, and adhesion without enlargement,"¶ and suggests that inflammatory engagement of the membrane only, or of the membrane and the muscular substance conjointly, may explain the difference. I believe that when the muscular substance of the heart has been seriously implicated in the inflammatory process, whether pri-

\* *Transactions of Medico-Chirurgical Society of Edinburgh*, vol. i., 1821.

† *Library of Medicine*, vol. v., p. 195.

‡ *Opus citat.*

§ *Opus citat.*

|| *Medico-Chirurgical Transactions*, vol. xxxv.

¶ *Loco citat.*

mary or secondary, degeneration and atrophy of the organ, at a more or less remote period, are the necessary consequence.

Wilks declares\* "that simple pericardial adhesions produce no visible ill consequences;" adding, "the loose cellular adhesions have no appreciable influence on the action of the heart, but that the thickened pericardium of cartilaginous consistency, investing the heart closely, arising from an inflammation at an early period of childhood, does lead to obstruction of the circulation, and then to dropsy, after the ordinary manner of heart-disease." He holds, also, that concurrent myocarditis may be the cause of subsequent dilatation of the heart by softening, the adhesions may involve the great vessels within the pericardium, and cause constriction of these, and its usual sequelæ; and thickening, he alleges, may extend into the cellular tissue of the anterior mediastinum and neck, and press on the veins, thereby causing cyanosis of the face, neck, and upper extremities.

The following cases (given in abstract) are published by Wilks in confirmation of the foregoing opinions:

Case 1. A man, aged twenty-one, ailing and short-breathed from childhood, subject to epistaxis and swelling of the feet, with lividity of the surface. No *bruit*. On dissection, universal adhesion of the pericardium, with bony deposit on the right side was found; enlargement of the auricles, but thinning and contraction of the ventricles; muscular tissue of the heart pale and flabby; valves healthy.

Case 2. A boy, aged nine, had scarlatina three years previously. Now livid, with dyspnœa and general dropsy. No *bruit*. P. M. General adhesion of the pericardium, which was as thick as leather from deposit of successive layers of false membrane, of which the internal were softer; the left ventricle was small and thin; the right chambers dense by infiltration of fibrinous tissue; valves healthy.

Case 3. A boy of twelve; some months before was attacked with dyspnœa after exposure; general dropsy gradually set in, accompanied by lividity; there was enlargement of the liver, but no cardiac *bruit*. P. M. General adhesion of pericardium, strongest and thickest on right side; heart normal as to size;

\* *Guy's Hospital Reports*, third series, vol. xiv., March, 1871.

valves healthy; liver of "nutmeg" character; lungs adherent to chest and to pericardium.

Case 4. A girl of sixteen, had rheumatism in childhood, since when symptoms of heart-affection had manifested themselves; died suddenly of pulmonary congestion and pneumonia. P. M. Lungs universally adherent to chest and to pericardium; latter attached to heart by false membrane, most firmly to right ventricle; all the chambers of the heart were thin except the left auricle; muscular substance pale and flabby; valves healthy and competent; mitral slightly thickened but competent.

Case 5. A man, aged thirty-six, had been in hospital a year previously for an affection of the heart, which yielded no abnormal sign except obscurity of the sounds; re-admitted with extreme dyspnœa and palpitation; there was slight dropsical effusion, but no *bruit*. P. M. Pericardium universally adherent, one-fourth of an inch thick, and indurated; heart generally enlarged; valves healthy; liver "nutmeg"; lungs generally attached to chest and pericardium.

Case 6. A man, aged twenty-four, had rheumatism some years previously, since when he suffered from dyspnœa, cough, and expectoration; after recent exposure dyspnœa became extreme, and was accompanied by lividity; no cardiac *bruit*. P. M. Lungs congested and universally adherent to chest and pericardium, which was firmly attached by false membrane to heart, and thickened; left ventricle normal; right ventricle hypertrophied; valves healthy, except aortic, which presented a few vegetations, but were competent.

The cases above narrated certainly afford no support to the opinion, that simple adhesion of the pericardium is competent to produce hypertrophy of the heart; nor do they in a greater degree favour the doctrine that atrophy of the heart is the normal consequence of this pathological change. Indeed, the conclusion to which they lead is rather of a negative kind, namely, that cohesion of the opposed surfaces of the pericardium has no positive influence as such upon the volume of the heart, and that when, in association with it, hypertrophy or atrophy of that organ is met with, other, and it may be, latent causes, are in operation to explain the connexion. Amongst the causes of

hypertrophy, valvular disease holds the foremost place; but, of this cause, dilatation of the chamber *behind* the affected valve in the course of the circulation, is always a concomitant result.

Chronic renal disease holds the next place in the order of frequency; but, owing partly to the occasional absence of prominent symptoms having reference to the kidneys, and, in some measure, likewise, to inattention to the state of these organs, the association of renal disease with cardiac hypertrophy has been frequently lost sight of. Disease or tissue-degeneration of the coats of the arteries, especially the ascending portion of the aortic arch, by imposing additional propulsion-duty upon the left ventricle, may be, and frequently is, a cause of hypertrophy of that chamber. Such hypertrophy, however, will be found to be simple, or unassociated with dilatation, unless softening or tissue-degeneration of the heart be a concomitant lesion.

I believe, as already stated, that pericarditis occurring in early life when the growth of the heart is in active progress, and involving only surface-irritation of the organ, whether arising directly from the inflammatory process, or, mediately, through the accumulated products of inflammation, and interfering in no degree with the coronary circulation, leads directly to general hypertrophy of the heart. I further hold that it will, of necessity, be followed by hypertrophy, provided only the patient survive for a period of, at least, one to two years, and that no countervailing agency come into operation, such as phthisis, or marasmus from any other cause.

Under the opposite conditions, namely, constriction of the heart and coronary arteries by exuded and organized fibrin, to such a degree that the circulation and tissue-nutrition of the organ is seriously impaired, the growth of the heart will be arrested in the child, and early death from failure of the circulation will be the necessary consequence. In the adult the same result will follow, but at a more remote period from the date of invasion, and as a consequence of actual and progressive atrophy of the heart.

Between the two conditions above sketched, there is conceivably one of an intermediate grade, in which the inflammation is neither sufficiently active to stimulate the heart to abnormal



excitement, nor its products in sufficient quantity to cause mechanical irritation or disturbance of circulation. In such cases, of which examples will be presented in the sequel, the heart will be found to have undergone no alteration of volume whatever.

If inflammation have, at the same time, extended to the substance of the heart, and caused softening of the organ, dilatation will be the result, with or without hypertrophy, according to the extent to which the nutrition of the heart, or its nutrient supply, has been compromised by the preceding inflammation. I regard external adhesion of the pericardium, whether to the lungs or to the sternum, as of much less consequence than tissue-softening of the heart, in determining dilatation of its chambers. The cooperation of both these causes, as can be readily conceived, will give rise to maximum dilatation, usually with hypertrophy, general or partial, because of the concurrent stimulus to nutrition. Where, with such a combination of organic lesions, produced in early youth and associated with valvular disease, life has been long protracted, an example of the *cor bovinum* or ox-heart is likely to be the result. A morbid specimen of this character, and exemplifying in the highest degree the alterations of volume and capacity in the heart which these causes are capable of producing, was exhibited to the Pathological Society of Dublin, by Dr. Stokes, in January, 1869.\*

Exuded lymph, the product of acute inflammation, if not already organized, is liable to absorption on the pericardial as on all other serous surfaces; but the membrane remains permanently opaque by interstitial cell-proliferation. It must be admitted, however, that organization and persistency, with or without adhesion, of lymph effused on the surface of the pericardium as the result of acute inflammation, is the almost universal rule; and, further, that of the cases in which lymph so produced is organized, ninety per cent., at least, furnish examples of mutual adhesion of the serous surfaces.

Mayne was of opinion that complete resolution may take place in the second stage of pericarditis, irrespectively of the extent of surface engaged.†

\* See Table II., Case 22, p. 358.

† *Dublin Journal of Medical and Chemical Science*, vol. vii., 1835.

Such was likewise the opinion of Kirkes.\*

Fuller holds that pericarditis of limited extent and mild character, may undergo complete resolution in the second stage, leaving only "white spots" as the traces of its former existence;† whilst Gairdner denies the resolution of acute pericarditis without adhesion.‡

The exuded lymph is of a pale rather than a blanched hue; it usually presents the appearance, at first, of disseminated, semi-liquid, minute droplets, which, after a period of twelve to twenty-four hours, become solid, and usually, likewise, dotted with sanguineous *puncta*. In very acute cases, the exuded fibrin is from the first of a blood-red tint. The opposed surfaces are, in most cases, coextensively, but not equally, affected, and the exudation may be universally diffused, or limited to one or more distinct and separate portions of the pericardial surfaces. In the latter case, the surface of the right auricle, the anterior surface of the right ventricle, and the right and left apex of the heart, to a variable extent, are the parts most usually affected.

Bouillaud has described, under the designation of "pine-apple heart," a condition of that organ, in which the changes consequent on pericarditis are presented in an exaggerated form. The heart is hypertrophied, and its entire surface is covered with minute conical masses of solid and organized lymph, resembling thus a pine-apple in appearance, no adhesion between the visceral and parietal pericardium having taken place.

In limited or circumscribed adhesion of the pericardium, the union of the surfaces is usually effected by bands of lymph, sometimes an inch or more in length, extending between them, and, but very rarely, by direct agglutination; whereas, universal adhesion is effected almost exclusively by close and direct union of the surfaces. In the former case it would seem as if the points of attachment were not sufficiently numerous to restrain the free movements of the heart, by which, consequently, the plastic lymph is drawn out in the form of filaments or bands,

\* *Medical Gazette*, 1849.

† *Diseases of the Chest*, 1862.

‡ *Monthly Journal of Medical Science*, February, 1851.

corresponding in length to the entire, or to a certain proportion, of the range of relative displacement of the surfaces, according to the degree of strength or capacity for resistance which they respectively possess.

It is scarcely conceivable that adhesion of this character can exercise any palpable influence upon the volume of the heart. I have never found, in connexion with it, either hypertrophy or atrophy of the organ, which was not susceptible of other and more satisfactory explanation.

General adhesion is effected by the mutual apposition and fixation of the two serous surfaces, through the medium of plastic lymph, which passes through the successive stages of vascularization, solidification, and contraction. The effused lymph at first opposes no obstacle to the free movement of the two surfaces, and soon acquires sufficient consistence to yield a friction-sound. When first heard, this sound is faint, and not specially harsh; it soon, however, acquires this quality, and in a ratio as to time and intensity proportionate to the increase of consistence of the lymph. As adhesion proceeds, owing to the increasing impediment to the free movements of the heart, the sound of friction becomes less and less representative of the rhythm of these movements; whilst, at the same time, it assumes a still more harsh and grating character, in consequence of the increasing density of the exudation; ultimately, a single sound of systolic rhythm, and usually basic in site, corresponding to the more powerful movement of the heart, is alone audible. Adhesion may be now regarded as all but complete; and at this period the uniting medium will be found condensed, contracted, and vascular.

In a few exceptional cases *frottement* has continued audible for several consecutive months over a very limited area, whilst in all other situations it had long previously ceased (case of the girl Young). Presumably, in these cases, adhesion had been completed at all points, save that at which friction-sound was still audible; but why the process of adhesion should have failed in this situation, and, as may be inferred from the existence of distinct friction-sound, without the intervention of liquid or gas by which the surfaces might have been kept apart, I am unable

to say. It occasionally happens that lymph of a pasty and aplastic character, is found smeared over portions of the heart's surface to which it loosely adheres. In such cases, pericarditis usually complicates general adynamic disease of some kind, such as typhoid pneumonia, typhus or typhoid fever, the low forms of scarlatina and variola, puerperal fever, phlebitis, delirium tremens, or pyæmia; and adhesion entirely fails. Detached flocks of pale fibrin are likewise, in such cases, not uncommonly found floating in the turbid serous contents of the pericardium.

I have not met with an example of typhus complicated with pericarditis, and Dr. Stokes has witnessed only one such case.\* When pericarditis occurs in connexion with any of the diseases characterized by the typhoid condition, I should expect that it would be *latent* as to symptoms; but even in such cases the physical signs are singularly constant.

On this subject Andral remarks:† “In the several cases which we have reported, pericarditis was announced by an assemblage of symptoms which rendered its diagnosis sufficiently easy.

“We are now about to adduce cases in which the most prominent of these symptoms, pain, did not exist; and it was only in some degree, by proceeding by exclusion, that one could arrive at a recognition of inflammation of the pericardium.”

The cases alluded to were examples of latent pericarditis; and the remarks bearing on them, just quoted, show clearly that Andral, at the date (1829) of issue of the second edition of his great work, from which the extract is made, was unacquainted with the pathognomonic sign of pericarditis.

Fuller truly remarks‡ that rheumatic pericarditis is seldom latent, whilst that of the non-rheumatic kind is often so.

Sir T. Watson gives the particulars of a very interesting case of latent pericarditis, illustrative of the features, negative and positive, of that form of the disease.§

According to Dr. Stokes, pericarditis, when latent, is invariably

\* *Opus citat.*, p. 80.

† *Clin. Méd.*, vol. i., p. 23.

‡ *Diseases of the Chest*, p. 513, *et sequent.*

§ *London Medical Gazette*, vol. xvi., part i., April 11th, 1835.



of the dry form, and that the more urgent symptoms by which the disease is usually characterized, are due, not to the inflammation of the serous membrane, but to one of its consequences, namely, serous effusion into the sac.

In the majority of cases of acute pericarditis, the plastic material constituting false membranes, connecting bands, or loose shreds found in the pericardium, would be correctly designated as a *deposit* from a liquid exudation identical with the liquor sanguinis.

Rindfleisch, however, states that the product of acute inflammation of serous membranes is twofold; a fibrinous deposit from the liquor sanguinis, as already described, which adapts itself to the various irregularities of the surface, forming a cast of it, without being organically united with it, and ultimately disappearing by fatty degeneration, liquefaction, and absorption, and very rarely undergoing caseation and no further change. Beneath this the epithelium is tumefied, its nuclei proliferate and burst from their respective particles, the *débris* of which is floated away in the general liquid contents of the cavity, to undergo ultimate solution and absorption; whilst the nuclei, greatly multiplied, enter into the formation of the subjacent plastic and organizable stratum. Of this latter element, however, the great bulk consists of embryonic connective tissue, derived from the proliferating connective tissue corpuscles of the subserous structure. This becomes organized, blood-vessels are formed in it by linear apposition and tunneling of cells, and by entrance of blood from neighbouring blood-vessels. Finally, the opposed surfaces come into contact, and are united by continuity of structure, and extension of blood-vessels from the one to the other through the connecting media; the embryonic tissue becomes fibrous, and contracts, vascularity is diminished, absorption of all unorganized material is completed, and solid permanent union is the result.\*

It occasionally, however, but rarely, happens, that plastic lymph alone is effused upon the serous surfaces, constituting "dry" pericarditis. It likewise occasionally happens that upon the serous surfaces fibrin is primarily effused, from which, in

\* *Pathological Histology*, Sydenham Society's edition, vol. i., p. 305.

process of organization, serum is subsequently expressed, and trickling to the most dependent portion of the sac, constitutes a veritable but limited serous effusion. It may be a question whether, in the majority of instances, this is not the mode in which primary serous collections of limited amount, and coeval with initial *frottement*, are formed.

In ordinary cases, therefore, *frottement* indicates not only the presence of lymph, of which it is directly symptomatic, but likewise that of its usual concomitant, serum. In the early stage of the affection, the latter is present in comparatively small quantity in most instances, and insufficient to prevent mutual contact of the serous surfaces at one or more points of their extent; it gravitates to the dependent portion of the sac, according to the posture of the body. Hence, when the patient lies upon the back *frottement* may be heard over the entire superficial precordium, and in the sitting posture only in the upper part, to an extent inversely proportionate to the height of the liquid level within. If the patient lie upon the abdomen, friction-sound may be heard, extensively in the left infra-scapular region, save in those cases, comparatively numerous, in which the posterior portion of the pericardium happens to be unaffected by the inflammatory process.

Doctor Latham maintains, but I cannot agree with him, that the exocardial murmur is "neither abolished, nor abated, nor otherwise altered in its character by the serum effused within its cavity."\* In the absolute sense in which this statement is intended to be applied, it is certainly incorrect; but that serum in small quantity is usually present in all cases save those of "dry" pericarditis, is no less certain. Hence, in great measure, the modification of *frottement* to which change of posture is known to give rise.

In cases involving difficulty of diagnosis, as between pericardial friction-sound and endocardial murmur, Sir D. Corrigan assigns great value to the evidence, positive or negative, afforded by the patient's assuming the prone or stooping posture.† If the sound cease to be heard when the patient sits up, and leans for-

\* *Lectures*, vol. i., p. 130.

† *Reports of Dublin Pathological Society*, vol. ii., December 7th, 1850.

ward, he would regard it as exocardial in origin, and due to pericarditis; and if still audible in that posture, he would regard it as endocardial.

This rule, though undoubtedly useful in the process *par exclusion*, and capable of yielding valuable aid in a certain percentage of difficult cases, is of only limited application, and involves at least two fallacies. Thus, in dry pericarditis, the pseudo-murmur may be even intensified by assumption of the stooping posture; and by the effort involved in assuming that posture, it occasionally, though rarely happens, that an endocardial murmur is masked or even suspended. This is especially true as applied to functional mitral murmur. (See pp. 285 and 287.)

Latham holds that cessation of *frottement* in pericarditis, unassociated with the signs of liquid effusion, implies, in all cases, without exception, cohesion of the surfaces of the pericardium. Yet, with marked inconsistency he maintains that cessation of an endocardial murmur would indicate resolution or absorption of the exuded fibrin.

I cannot admit either proposition absolutely. Pericardial *frottement* may be finally suspended without adhesion, when it has been originally due to the presence of aplastic fibrin, in connexion with any of the various forms of general adynamic disease. In such cases, whilst adhesion fails from want of organizing energy in the tissues and the recent exudation, *frottement* is not uncommonly suspended or abolished, owing to typhoid softening and impaired contractile energy of the heart. In the second place, inasmuch as endocardial murmur, associated or not with pericarditis, may be, and frequently is, due to other causes than endocarditis, as already stated, for example, temporary debility, and local yielding of the walls of the left ventricle during systole, the cessation of it cannot be accepted as proof of the absorption of the products of endocarditis.

Serous effusion may increase, and usually does so, to the extent of completely separating the surfaces of the pericardium, and suspending friction-sound and *frémissement*. When this has occurred, the third stage, so called, of pericarditis, or that of liquid effusion, has been established; but this stage implies only the increase of an effusion which had previously existed. On

the other hand, instead of increasing, it may undergo decrease, and the surfaces, being now in extensive and uninterrupted contact, will become united through the medium of organized lymph previously deposited upon, or exuded from, the serous surfaces. If the lymph effused be of a low type, or aplastic in character, it may remain unorganized, and either feebly attached to the surface of the pericardium, or as loose shreds within its cavity, adhesion having in no degree taken place.

Such aplastic exudations are of usual occurrence in the pericarditis which complicates chronic renal disease and adynamic fevers of a septic character. They are in process of slow fatty disintegration. In chronic pericarditis generally, which is usually of non-rheumatic origin, there is, as justly maintained by Laennec, rarely a false membrane; when such is present, it is soft, friable, and like a layer of thick pus, and accompanied by a turbid puriform liquid effusion. This form of the disease is, he alleges, always general, engaging the entire surface of the pericardium; the exuded lymph is likewise redder than in the acute form, the redness being due to many minute blood-points. When pericarditis arises from, or is associated with, diffuse inflammation, then effusion into the pericardium is likely to be purulent.\*

In connexion with acute pericarditis, blood is occasionally found, even in considerable quantity, in the sac of the pericardium. An example of this kind is mentioned by Dr. Latham.†

Andral furnishes a still more striking example. A man, aged thirty-one years, was attacked with severe radiating and burning pain in the region of the heart, followed by numbness extending down the front of the left arm and forearm, and alternating with excruciating pain in the same parts, during which respiration became greatly oppressed, the heart's action tumultuous and irregular, pulse failing, and surface cold. After a few minutes, pain ceased and numbness returned; the pulse was now regular but small; breathing less embarrassed, but the action of the heart still quick and energetic, and heard over the entire front of the chest.

\* See a case by Dr. Law : *Reports of Pathological Society of Dublin*, vol. i., p. 42.

† *London Medical Gazette*, vol. iii., p. 7, December 6th, 1828.



On the fifth day of illness the patient was convalescent; but, on the following day great weakness and symptoms of sinking suddenly appeared; the heart's impulse was no longer perceptible; the precordium was extensively dull, and the patient died in the course of the night. About two pounds of blood were found in the pericardium, and shreds of false membrane covering its surface.\*

In such cases the diathesis is hæmorrhagic, and a state of hypnosis, or blood-attenuation, exists; evidence of which may be found in the tumid state of the gums, and in the liability of the patient to bleeding from the mouth, nose, bladder, and intestinal canal, and to ecchymosis of the conjunctivæ after slight irritation.

Bouillaud truly remarks that simple blood-staining of the false membrane of pericarditis is not uncommon, and that it is most frequently the immediate result of imbibition from a blood-stained liquid effusion.

*Gaseous exhalation* into the pericardium, the direct result of inflammation of its serous lining, has been likewise observed.

Graves details the following example of pericardial pneumatosis. A girl, aged twenty-five, was suddenly attacked with severe and general pain in the abdomen, resembling peritonitis; five days subsequently a tumor of a conical shape, yielding distinct fluctuation, and dull on percussion, appeared at the epigastrium, extending downwards within two inches of the umbilicus, and into either hypochondrium; two days later purging suddenly set in; the epigastric tumor, now slightly reduced in size, no longer yielded fluctuation, and was tympanitic on percussion; purging continued; the patient became gradually weaker, and much wasted; the tumor had entirely subsided after an interval of twenty days from the date of its first appearance, when she was suddenly attacked with violent pain in the region of the heart, accompanied by palpitation, and a sensation of burning beneath the ensiform cartilage. The precordium was now found to be tympanitic on percussion; double *frottement* was heard over the base of the heart, and occasionally a metallic click inside the nipple; next day, emphysematous crackling

\* *Clinique Méd.*, second edition, 1829, vol. i., observ. iii., p. 15.

was heard, and after a further interval of two days, a regular metallic tinkling, synchronous with the heart's action, took the place of the latter phenomenon, and superseded all other physical signs, except a slight *bruit de soufflet* at the mamma. Death took place one month from the date of first illness. A large abscess was found in the left lobe of the liver, communicating with the stomach by three several openings, and with the pericardium by another through the diaphragm. The pericardium was inflamed, thickened, coated with lymph, and contained about two ounces of pus.\*

The pericardium in this case became inflated from the stomach, whilst the pus found in it proceeded from the hepatic abscess, and was the immediate cause of pericarditis.

Doctor Stokes gives a case of primary pneumatosis of the pericardium, so interesting that it deserves to be reproduced in its entirety. In his own words, "A young man, of lymphatic temperament, had laboured under an attack of acute pericarditis for a few days before I saw him. On my first examination, he presented the usual signs of dry pericarditis, with a considerable effusion of lymph of the ordinary consistence. The rubbing sounds, though loud and distinct, had nothing unusual in their character, and the patient suffered but little distress. After two or three days I saw him again, and found that his state had become very much altered. His appearance was haggard and worn, and he complained of extreme exhaustion, which he attributed to a total deprivation of sleep. This was induced by the extraordinary loudness and singular character of the sounds proceeding from the cardiac region; for though up to this period the rubbing sounds were distinctly perceptible by means of the stethoscope, the patient was quite unconscious of their existence. They had suddenly, however, become so loud and singular, that the patient and his wife, who occupied the same apartment, were unable to obtain a moment's repose. On examination, a series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sounds of indurated lymph, or the

\* *Clinical Medicine*, vol. ii., p. 233.

leather creak of Collin, nor those proceeding from pericarditis with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met so extraordinary a combination of sounds. The stomach was not distended by air, and the lung and pleura were unaffected, but the region of the heart gave a *tympanitic bruit de pot fêlé* on percussion, and I could form no conclusion but that the pericardium contained air, in addition to an effusion of serum and coagulable lymph.”\*

I have not met with an example of this singular condition, but judging from those above given, I conclude that it would be distinguished by the following peculiarities. 1st. Precordial tympany without displacement of the heart, and distinguishable from that of the stomach, by the *absence*, higher up, of normal precordial dulness. Pulmonary emphysema, would, however, modify the last mentioned diagnostic, but this may be readily distinguished by its proper signs, especially the *downward* displacement of the heart, a change of position the reverse of that which the heart would assume from the pressure of an inflated stomach. 2nd. Pericardial *frottement* of extraordinary intensity, and audible to the patient and to bystanders. 3rd, but of less diagnostic significance than the former, an overwhelming sensation of oppression and sinking,

Whatever the nature of the pericardial effusion be, whether serum, pus, blood, or gas, it may prove rapidly fatal by impeding the action of the heart, either by its absolute quantity, or by still further embarrassing an already feeble organ. It may, on the other hand, undergo absorption, and its liquid portions be quickly and entirely removed, leaving, in the case of purulent or sanguineous effusion, a solid deposit upon the serous surfaces. This deposit is paste-like, pale yellow in colour, and of a granular and non-coherent texture where the antecedent effusion has been purulent; but, where blood has been extravasated, the residuum, after absorption of its liquid portion, consists of fibrin and the *débris* of the blood-corpuscles, tinted in various shades of red, according to the time elapsed since the primary effusion.

\* *The Diseases of the Heart and the Aorta*, 1845, p. 21.

In recent cases the deposit presents the decided tint of blood; and, after a protracted interval, during which it has been subjected to a process of gradual absorption, the colouring matter may be entirely removed, and the new surface may be found of a colour only a shade deeper than that of a primary exudation of fibrin. Extravasation of blood into the pericardium, of non-traumatic origin, is usually consecutive to inflammation, and of much later date; indeed, its most frequent source is the vascular plexus developed in the recently organized lymph upon the serous surfaces. Hence, the appearance resembling the granulated surface of a healing ulcer, to which, when examined at an early period, it has been aptly compared; or where the subjacent exudation of lymph has assumed the character of conical growths, and become solid in texture, the surface presents the appearance of the dorsum of a neat's tongue smeared with blood. The term *hæmorrhagic* is used to designate this form of pericarditis, which is usually the consequence of an *accident* of ordinary serous inflammation, occurring in a diathesis favourable to sanguineous effusion. Rokitansky declares that it is of most frequent occurrence in connexion with tuberculous pericarditis. Examples of sanguineous and purulent collections in the sac of the pericardium will be found recorded in Tables I. and II. (See pp. 350 and 354.)

The title *dry pericarditis* is intended to designate a form of the disease in which the morbid process is arrested in the second stage, and liquid effusion does not take place. It is usually, at the same time, slow as to progress, and latent as to symptoms.

Doctor Stokes declares that he has rarely met with it where pericarditis was not complicated by "inflammation in various organs and different tissues."\* In most of the cases which have come under my notice there was preexisting renal disease, with hypertrophy of the heart, and the friction was characterized by extreme loudness, extensive diffusion, and long continuance. Indeed, I cannot recall an example of this form of pericarditis which proved fatal, in which the friction-sound did not continue up to a brief period preceding death, when failure of the heart was the cause of its cessation.

\* *The Diseases of the Heart and the Aorta*, p. 66.



Doctor Stokes has noticed its continuance in one case for a period of sixteen days. Examples of no less duration will be found amongst the cases appended to this chapter.

I have further noticed that in all these cases the primary disease, of which pericarditis was a complication, had already given rise to dropsical effusion elsewhere, usually of the feet and legs, previously to the occurrence of pericarditis. Has this effusion exercised a *derivative* influence upon the pericardium in process of inflammation? Judging from analogy, I am disposed to answer in the affirmative. The chronic character of the disease, and the absence of liquid effusion in these cases, may likewise serve to explain the ill pronounced character of the symptoms, or latency of the affection. The intensity of the *frottement* may be accounted for in part by the total absence of liquid from the sac, but chiefly by the coexistence of hypertrophy of the heart. The physical signs distinctive of dry pericarditis are persistent clearness on percussion beyond the normal area of precordial dulness, distinct *frémissement*, and loud, long-continued, and extensively diffused friction-sound. Judged by its associations, it is of most unfavourable augury.

In a few examples already adverted to, I have met with dry pericarditis under different circumstances, and with a very different result. The patients were affected with rheumatism of a mild character, and exhibited no cardiac symptoms whatever. Nevertheless, circumscribed basic *frottement* existed, continued unaltered through convalescence, and, at the end of several weeks, when the patients were discharged "cured," was still present and no less distinctly pronounced. I regard this as due to permanent roughening of a limited extent of the pericardial surfaces by lymph deposited in a granular form, and arrested in its process of organization at the stage in which its vascular supply sufficed barely for the maintenance of its vitality, and was incapable of establishing adhesion by extending into contiguous tissue. So far as I know, friction-sound of this kind is permanent, and the condition which causes it leads to no alteration in the volume or structure of the heart, and in no degree shortens life.

*Hydro-pericardium*, or collection of serum in the pericardial

sac, would mean, according to the definition of Corvisart, all such accumulations exceeding in quantity six ounces. He mentions a case in which eight pounds of serum were found in the pericardium. The quantity, however, rarely exceeds two pints. After death a few drachms of serum are always to be found in the pericardium, even when death has surprised the party in a state of perfect health. This is the result of *post mortem* condensation of the halitus present in the pericardium, as in all serous cavities, during life; the source of which is the epithelial surface, and the purpose to be served, that of facilitating the gliding movements of the opposed surfaces upon one another in the process of "functioning" of the invested viscera. The accumulation of a larger quantity is morbid, and the result of derangement of the balance between secretion and absorption. It is not difficult to conceive how such derangement may arise in connexion with inflammation, associated as that process is with vascular engorgement, and deranged vascular pressure and tension. If, at the same time, owing to attendant pyrexia, impaired secretory function of other organs normally engaged in the elimination of water from the body be a concomitant symptom, as is usually the case, excessive secretion of serum into the pericardium, and absolute, as well as relative, retardation of absorption, must be the result.

The diagnosis of liquid effusion in the pericardium, when in small or even moderate quantity, is usually attended with considerable difficulty.

Galen\* held that in such cases the palpitation of the heart communicates a sensation as if the organ were moving in a fluid. He also mentioned, as symptomatic of hydro-pericardium, great languor, drooping of the eyes and somnolence, palor and lividity of the lips and finger-ends, great loss of flesh, weak, small and intermitting pulse, and dyspnoea, with extension of the neck.

Morgagni mentioned only two symptoms as possessing diagnostic value; namely, a sense of weight on the heart, and of squeezing oppression at the precordium on making exertion. In the case of the virgin recorded by him,† the colour of the face was good, sleep undisturbed, bowels and menstruation regular,

\* Quoted by Morgagni.

† *Epistola* xvi.

breathing equally easy in the standing, supine, and in either lateral posture, pulse natural, and no palpitation, pain in chest, or cough. He denied that swooning and cough are usual.

Senac\* speaks of dropsy of the pericardium as a disease "that is frequent, difficult to be known, and more difficult to be cured."

Corvisart regarded, as the symptoms of hydro-pericardium, lividity of countenance, anxiety and sense of weight, and tumultuous pulsation at the precordium, great dyspnœa in the horizontal posture, syncope, palpitation, small, feeble, frequent, and occasionally irregular pulse, great debility, extended dulness, often protuberance of the left side, œdema of lower limbs, and occasionally puffing of the front and left side of the chest.

He once felt, but never saw, localized fluctuation in the fourth and fifth intercostal spaces, as mentioned by Senac. He attached some diagnostic value to threatened suffocation from lying on the right side, and, still more, to the sign which he claimed to have discovered; namely, pulsation of the heart at various and distant points, owing, as he believed, to the floating of the organ in a liquid medium, and within an enlarged pericardium, as determined by the movements and posture of the body.

Doctor Latham says that where effusion into the pericardium coexists with exocardial murmur, which, according to him, is usually in no degree modified by it,† an undulatory movement in the third and fourth left interchondral spaces may be seen, and vibration felt in the same situation. He adds that vibration is more frequent, and often unaccompanied by undulation, but undulation never without vibration.

All these alleged symptoms of effusion into the pericardium are eminently equivocal, with the exception of precordial dulness,

\* Lib. iv., ch. v.

† Andral (*Anatomie Pathologique*, p. 520, foot note) mentions a case in which two and a-half pints of liquid were found in the pericardium, yet friction-sound continued up to the time of death. In this case, however, there was considerable hypertrophy of the heart, and the usual posture of the patient was that of stooping, the elbows resting on the knees. It is possible, therefore, as nothing is stated to the contrary, that partial adhesion may have taken place between the heart and anterior wall of the pericardium. Again, it is not at all improbable that increase of the effusion took place in the death-struggle, and after the final examination of the patient's chest, as frequently happens.

and localized precordial œdema and protrusion. Even these symptoms are not conclusive, but they are much more significant than the others included in the preceding category.

The symptoms and signs of liquid effusion into the pericardium may be classified under two heads; namely, equivocal and unequivocal. Under the head of signs I propose to include only the evidence which may be elicited by auscultation and percussion.

Of the preceding list of equivocal symptoms, to which should be added dysphagia, I will discuss here only that observed by Senac, and subsequently sanctioned by the authority of Corvisart and Latham; namely, intercostal fluctuation and dysphagia.

I have never witnessed intercostal fluctuation in connexion with hydro-pericardium, and, therefore, I have no personal knowledge of the value which should be assigned to this alleged symptom. Judging, however, from my general knowledge of allied morbid conditions, I incline to the conclusion that this symptom will be manifested only where the intercostal muscles are paralyzed and protruded;\* and as this is most likely to be the case in pyo-pericardium, it becomes a question whether intercostal fluctuation, limited to the precordium, should not be regarded as pathognomonic of purulent collections in the pericardium.

Dysphagia has been noticed by Stokes, Mayne, and others, in connexion with liquid accumulations of large amount in the pericardium.

It is conceivable on anatomical grounds, that from such a cause difficulty in the deglutition of solids may arise; but, as a matter of fact, such difficulty has been noticed by the most competent observers. Viewed in the abstract, however, it cannot be regarded as possessing much diagnostic significance; because, in the first place, it may be due to morbid changes within the œsophagus itself, either accompanied by, or independent of, pericardial effusion; or to the pressure of aneurismal, cancerous, or other tumors in the posterior mediastinum; and, secondly, because most assuredly many examples, I would say, indeed,

\* See Dr. Stokes on the "Signs of Empyema," *Dublin Medical Journal*, vol. ix.



the greater number, of copious effusion into the pericardium, are unaccompanied by this symptom.

If, on the other hand, by the history of the case, these possible causes of dysphagia, intrinsic and extrinsic, be excluded, and pericardial effusion be, on other grounds, presumably present, then the coexistence of dysphagia assumes much corroborative value. When present, dysphagia is usually associated with weak and high-pitched voice; but it may occur in the first stage of pericarditis complicated with diaphragmatic pleurisy, in which case it would not, of necessity, be accompanied by dysphonia. Amongst the unequivocal symptoms of copious effusion into the pericardium, I would place precordial enlargement, determined by measurement taken from the posterior fold of the axilla to the mesial line of the sternum on each side, whilst the arm is raised above the horizontal line, and the hand rests upon the head. The existence of relative enlargement, so determined, will possess more significance if presented on the side of the body on which the arm is less freely used, because of the possible error arising from excessive muscular development. It will appear in the sequel, that if this phenomenon coincide with certain physical signs, to be mentioned presently, the diagnosis of pericardial effusion of large amount may be confidently made.

The physical signs of effusion which are of equivocal value are negative and positive :

*Negative.*

1. Absence of respiratory sound to an abnormal extent in the precordium.
2. Absence of cardiac sounds of normal distinctness.
3. Absence of, or very feeble cardiac impulse.

*Positive.*

1. Dulness on percussion limited to the precordium.
2. Displacement, temporary or permanent, of the point of apex-pulsation.
3. Friction-sound synchronous with the movements of the heart.
4. Disproportionate extension of dulness upwards.

The absence of respiratory sound over an increased precordial area, taken alone, possesses no special significance; because it may be due to tumor in the anterior mediastinum, cancer of the left lung, circumscribed pleuritic effusion or empyema in the anterior and internal portion of the left pleura, solidified aneuris-

mal tumor, abscess descending from the neck, or hypertrophy of the heart.

Partial suppression of the sounds of the heart may result from functional debility, or fatty degeneration of the organ; and from the use of certain medicinal agents in excessive doses; for example, bromide of potassium, digitalis, and nicotine. Impairment or suppression of the impulse may be the consequence of fatty transformation or extreme dilatation of the heart and thinning of its walls; and both the impulse and the sounds may be impaired, or apparently suppressed, by displacement of the heart from the anterior wall of the chest, through the agency of an emphysematous or a cancerous lung, or a mediastinal tumor.

Increase of the area of percussion-dulness in the precordium may be due to any of the causes above enumerated, as capable of giving rise to enlargement of the area over which respiratory sound is suppressed; to which may be added, localized pneumonic solidification of the anterior portion of the left lung.

Displacement of the apex of the heart may be caused by liquid or aeriform effusion into either pleura, enlargement of the liver, hepatic or perihepatic abscess, aneurism, mediastinal tumor, or diaphragmatic hernia of the stomach.

Pericardial *frottement* may be simulated by pleuritic friction-sound developed in the anterior portion of the left pleura; by an endocardial-murmur; and by emphysema of the anterior mediastinum, as shown by Dr. Hudson.\* It may, in every case where present, be distinguished from the two former phenomena; but it is wanting in most cases of liquid effusion into the pericardium to an amount capable of giving rise to morbid signs.

Da Costa maintains that suspension of sound during some of

\* *Dublin Quarterly Journal of Medicine*, vol. viii., p. 241, August 1st, 1849. In this interesting case there was extensive dulness of the precordium; but it does not appear that this was in any degree qualified, as might be expected, by mediastinal resonance. A *frottement* of a "crackling character," synchronous with the sounds of the heart, but strikingly distinct from them, and loudest to the left of the sternum, was detected at the base of the heart. The diagnosis of pericarditis was made by the author of the paper, a most accomplished stethoscopist. On examination of the body, the pericardium and heart were found to be perfectly sound; the areolar tissue of the anterior mediastinum was distended with air, and extended somewhat to the left, in front of the pericardium. A blood-clot contained in the right auricle was likewise partially occupied by air-bubbles.

the beats of the heart constitutes the only means of distinguishing pericardial from localized left pleural *frottement*.\* No doubt, occasional intermission of the friction-sound would warrant a *presumption* in favour of pleuritis; but much stronger evidence to the same effect might be deduced from the localization of the friction-sound *externally* to the apex-point, and its total absence elsewhere in the precordium, as pointed out by Dr. Stokes, independently of the coexistence of respiratory-sound in the seat of friction, where pleuritis is the cause of the phenomenon.

Extension of precordial dulness upwards, in a degree not readily accounted for by reference to other conceivable causes, should raise a suspicion of pericardial effusion. It may, however, depend upon pneumonia limited to the superior lobe of the left lung, carcinoma of the lung or mediastinum, or circumscribed empyema.

The elements for a positive diagnosis of liquid in the pericardium are of a compound character, and are rarely all present in any one case; hence the difficulty, in many instances, of arriving at a positive opinion.

Localized bulging of the precordium, engaging an increased extent of surface, dull on percussion, devoid of respiratory sound and movement, and of vocal thrill, without displacement of the heart, the cardiac sounds being faint and distant,† and associated or not with *frottement* and irregular action of the heart, might be held to warrant the diagnosis of liquid in the pericardium. Obliteration of the intercostal spaces in the precordium, with visible undulation synchronous with the movements of the heart, variation of the seat of apex-pulsation according to the posture of the body, and displacement of the liver downwards, should be regarded, when present in the same case, as confirmatory of the diagnosis of pericardial effusion. But, although of the utmost value diagnostically, these signs must not be looked for in the majority of such cases, because their presence is certainly the exception. I cannot say that I have

\* *Medical Diagnosis*, third edition, 1870, p. 357.

† Dr. Da Costa (*opus citat.*, p 355) justly remarks, that where the sounds of the heart are masked in the precordial region by liquid effusion into the pericardium, the second sound is distinct, and even sharp, along the sternum and in the course of the ascending aorta.

derived much assistance from the alleged triangular definition of the area of extended precordial dulness in these cases. Indeed, I believe this test to be practically unavailable, from the difficulty of tracing the outline of a dull surface of limited extent, with such accuracy as to distinguish between one of a triangular and one of an ovoid figure. It must be borne in mind, as eminently pertinent to this inquiry, that disproportionate extension of the horizontal line of precordial dulness inferiorly, which is alleged to be distinctive of effusion into the pericardium, may be due to enlargement of the liver or spleen.

Extension of localized dulness considerably to the left of the point of apex-pulsation, if associated with absence of respiratory sound of any kind to the same extent, would assist materially in establishing the identity of liquid effusion into the pericardium, and would suffice to distinguish it from hypertrophy of the left ventricle on the one hand, and from localized solidification of the adjacent portion of the left lung upon the other; and, similarly, limitation of dulness to the antero-inferior portion of the chest, equally in the erect and recumbent posture of the body, would practically exclude liquid effusion in the left pleural cavity.

Unfortunately for the value of the former of these alleged diagnostics, the apex-pulsation is *not* to be felt where the pericardium is distended with liquid, save in the few cases in which the apex is fixed by adhesion to the precordium. The heart may, no doubt, be brought into contact with the anterior wall of the chest by causing the patient to assume the prone or stooping posture; but in this position of the body it would be difficult, or even impossible, to determine the extent of dulness, with a degree of accuracy sufficient to constitute it a legitimate basis for differential diagnosis. Nor, indeed, does it seem necessary; for the distinction between left ventricular hypertrophy and hydro-pericardium, even based upon the positive signs of the former affection exclusively, can never be difficult.

Doctor Gee says, "The first extension of percussion-dulness occurs at the base of the heart, where the great vessels enter, and where the pericardium hangs loosely round them, and is most distensible."\* But the pericardium is in a state of ten-

\* *Auscultation and Percussion*, 1870, p. 247.



sion physiologically at all points. Under the influence of the inflammatory process, however, it becomes relaxed, and equally so, throughout its entire extent. As a matter of fact, I have never met with an example of primary dulness at the base of the heart from pericardial effusion, and cannot admit such, either on physiological or clinical grounds, till definite proof of its occurrence shall have been furnished. On the contrary, I have found that in the progress of adhesion or liquid effusion, persistence of friction-sound at the base, even for several days after it had ceased to be audible elsewhere, whether with or without increase in the area of precordial dulness, is the rule. Such is likewise the opinion of Da Costa, who says that when the stage of serous effusion has set in, and the pericardium contains liquid even in considerable quantity, "it is not uncommon for the ear still to recognize the murmur at the base of the heart, and around the origin of the great vessels."\*

*Recurrent pericarditis*, by which is meant a second or subsequent invasion of the disease, is characterized by a group of symptoms dependent upon the nature and extent, not only of the present, but likewise of the previous attack. If complete adhesion be amongst the consequences of the primary affection, the secondary accession will not be characterized by friction-sound or fremitus. Partial adhesion in a previous attack, however, does not preclude friction-phenomena in one of a later date, but at a point of surface distinct from that where adhesion has actually taken place.

The recurrence of such phenomena, therefore, amounts to proof that antecedent pericarditis has not issued in universal cohesion of the parietal and visceral pericardium. In such cases, also, liquid effusion can be only partial, and superficial dulness must be proportionately limited.

Doctor Moxon is of opinion, and I quite agree with him, that disorganization of the epithelium, which is the necessary result of adhesion of the pericardium, indisposes the serous structure to recurrent inflammation.† When such inflammation

\* *Opus citat.*, p. 353.

† "Lectures on Analytical Pathology;" lecture viii. *Medical Times and Gazette*, November 12th, 1870.

occurs, therefore, the cause may be presumed to have been of a more than ordinarily exciting character, and the results, both as to symptoms and inflammatory products, in excess of those usually witnessed. Hence the aggravated character of the symptoms, and the great thickness of the pericardium, by which recurrent pericarditis is generally characterized.

If, in a previous attack, the substance of the heart has been invaded by inflammation, and consecutive degeneration of its fibres has made progress in the interim, the symptoms of the recent accession will be of a formidable character *quoad* the circulatory and respiratory functions. In such a case there is likely to be failing pulse, vertigo, sensation of sinking and oppression at the precordium, palpitation and threatened syncope on making even the slightest effort, oppression of breathing, and congestion and cedema of the lower limbs. Epigastric fulness and tenderness, and slight albuminuria, but without lowered specific gravity of the urine, are amongst the consecutive but late symptoms of recurrent pericarditis, where tissue degeneration of the heart has preceded. Manifestly, in such a case, the heart would barely be competent to maintain the circulation at the standard of feeble health; hence, when further embarrassed by the products of renewed inflammation, or debilitated by a secondary extension of that process to its muscular substance, the heart is no longer capable of performing its functions, and the consequences of its partial but progressive failure are amongst the symptoms of the recurrent disease. Antecedent endocarditis, by disorganizing the valves, and thereby entailing upon the heart the consequences of valvular lesion, namely, hypertrophy and dilatation, may be likewise the indirect cause of a formidable group of symptoms having reference to partial failure of the circulation, in a primary or secondary attack of pericarditis. It is noteworthy that, whilst a second attack of endocarditis is of exceedingly rare occurrence, secondary pericarditis is by no means uncommon; and further, that the secondary disease is usually not different from the primary, either as to site or essential character, the apparent difference depending upon the reduced power of the heart by preceding inflammation.

Under the designation of *latent pericarditis*, Drs. Law and

Stokes have described a form of the affection which is not announced by symptoms ; the physical signs, however, being well pronounced.

Doctor Law has met with examples of it in connexion with disease of the lungs, both acute and chronic ;\* and Dr. Stokes has witnessed it as a complication of the essential fevers, and in diffuse inflammation, phlebitis, and erysipelas.†

In the preceding connexions, the symptoms of pericarditis are *masked* by the constitutional disturbance attendant upon a major disease.

*Chronic pericarditis*, according to Corvisart, is obscure in origin and difficult of diagnosis.‡ Laennec says it is always general, occupying the entire inner surface of the pericardium, and that the surface is of a redder tint than in the acute form, owing to the presence of a multitude of minute vascular points.§

It is most frequently the consequence of slow convalescence or protracted dissolution, from an attack of the disease in the acute or subacute form. It may, however, be primary, and in that case it is usually associated with chronic visceral disease, *par excellence*, that of the kidneys. When primary it is usually latent as to symptoms, being announced only by physical signs, which are, however, most commonly of an exaggerated character. In this form of the disease, likewise, the fibrin effused upon the surface of the pericardium rarely undergoes organization, remaining loosely attached to the surface, or floating in the liquid contents of the sac as white filamentous shreds. This is the form in which pericarditis is most frequently associated with hypertrophy of the heart, because of the concomitant disease of the kidneys to which both are due ; or with hypertrophy and dilatation, the consequence of valvular disease coeval with the pericarditis, and the direct source of the increase in the volume and capacity of the heart. The structural changes in the pericardium, constituting in such cases the first and the most striking

\* *Proceedings of the Pathological Society, passim.*

† *Diseases of the Heart and the Aorta*, p. 838.

‡ Hebb's translation.

§ *Mediate Auscultation*, Forbes' edition, p. 661.

alteration to be met with in a *post mortem* examination of the heart, and affording as they do, on the authority of Hope, a full and satisfactory explanation of the increased volume of that organ, have been admitted by pathologists as in every instance the primary lesion, and the starting point of the other organic changes discovered. The state of the kidneys has been rarely inquired into, and valvular disease of the heart, when detected, has been regarded as a contributory cause of hypertrophy, but of secondary importance in its influence.

Doctor Barclay states that in twenty-two out of sixty-one, instances of valve-disease, there was disease of the kidneys with albuminuria.\* No doubt, albuminuria is of frequent occurrence in valvular disease, indeed almost universal in the advanced stages; but it is in the majority of cases the result of simple congestion of the kidneys, the specific gravity of the urine being rarely under 1030.

*Circumscribed pericarditis*, as indicated by localized friction-sound, may be presented under two opposite conditions, as stated by Doctor Stokes;† one, in which, apparently, from failure or arrest of inflammatory action due to an unknown constitutional influence, or to early and effective treatment, the exudation of lymph is limited to one or more circumscribed spaces, over which for a variable, but sometimes for a considerable period, the sounds of friction may continue to be heard. In such cases the seat of friction usually corresponds to the sides of the ventricles, the most frequent site being, according to my experience, the third and fourth intercostal spaces of the left side, midway between the nipple-line and the sternum. In such cases, likewise, adhesion, or serous effusion rarely takes place. Secondly, a condition in which, after general pericarditis, and, it may be, liquid effusion, adhesion has proceeded to all but complete obliteration of the pericardial sac; in such case, friction may continue to be audible over a small space, generally at the base, but occasionally at the apex of the heart, for an indefinite period, and may mislead an incautious auscultator into making the diagnosis of hæmic murmur or valvular lesion.

\* *Medico-Chirurgical Transactions*, vol. xxxv., November, 1851.

† *Diseases of the Heart and Aorta*, p. 17.



The observance of the rules already laid down for the distinction of pericardial *frottement* from endocardial murmur, to which I beg to refer the reader, will always, however, render such a mistake in the highest degree unlikely to occur.

“*Milk spots*” are, I believe, due to circumscribed and aborted pericarditis of a subacute character, and identical with that form of arachnitis in which thickening and opacity of the membrane constitute the only visible alterations of texture resulting from even fatal inflammation; the epithelium is perfect in the true “milk spot.”

Corvisart denied the inflammatory origin of these patches, and attributed them to fibrinous deposit *beneath* the pericardium.\*

Laennec asserted that there was no example of albuminous exudation on the attached surface of a serous membrane; adding that pseudo-membranous exudations are almost invariably products of inflammation. He regarded the “white spots” as due to inflammation of the pericardium, and as consisting of deposit *on* its free surface.† Such cases I would regard as veritable examples of circumscribed and arrested pericarditis, yielding friction-sound during life, and presenting, when examined after death, a rough surface due to deposited fibrin, which, in many instances, may be removed from them in form of flakes. In neither of these particulars does the true white spot answer to the description just given; it is not associated with abnormal sound of any kind, and its surface is invariably smooth and glistening, and formed of tessellated epithelium.

Louis agreed with Laennec in regarding the “white spots” as the result of pericarditis, but limited to the visceral layer exclusively.‡

Mr. Paget says that in connexion with these spots there is usually, to some extent, adhesion of the pericardium; from which circumstance he concludes they are of pericarditic origin. In the greater number of the examples which have fallen under his notice, adhesion did not exist in the situation of the spots, a circumstance due, as he supposes, to the separa-

\* Hebb's translation, p. 40.

† *Mediate Auscultation*, by Forbes, p. 661.

‡ *Mémoires ou Recherches Anatomico-Pathologiques*, 1826.

tion of the surfaces by liquid during the progress of adhesion of the neighbouring surfaces. The adhesions consist of filaments between the aorta and superior cava, or the aorta and pulmonary artery; between one of these vessels and the parietal pericardium, or between one of the spots and the latter surface. Finally, there may be only vestiges of adhesion in the form of "small pearly granules." Their most common site is the anterior surface of the right ventricle; less commonly they are found on its posterior surface, or on the right auricle; still more rarely on the left ventricle; and least frequently on the left auricle. Their average width is half an inch; their thickness varies from half a line to two lines; and, according to the degree of inflammation, and the depth to which the membrane is engaged, the exuded lymph will occupy its surface or its substance. When the organization of the exuded lymph is complete, a new epithelium is formed upon its surface.\*

In forty-five, out of one hundred and fifty-six bodies, Bizot has observed the "white spot" on the right ventricle. He regarded it as due to thickening of the pericardium from some unknown cause. In his opinion it is three times more common in males than in females; rarely, if ever, seen before the age of seventeen; and not common before forty years.†

Doctor John Taylor has found "white spots," or patches, in a nearly similar proportion of the bodies examined in University College Hospital; viz., in one out of every four.‡

Finally, Copland attributes the "white spots" to pericarditis.§

Thus, it would seem to be the opinion of the majority of writers, that the patches usually seen upon the heart in advanced age, and designated "milk spots," or "white spots," are due to antecedent inflammation.

In this opinion, however, as above stated, I by no means share.

*Endo-pericarditis*, as may be inferred from what has preceded,

\* *Medico-Chirurgical Transactions*, vol. xxiii., 1840.

† *Recherches sur le Cœur et le Système Arteriel chez l'Homme*.

‡ *London Medical Gazette*, new series, vol. i., 1845.

§ *Dictionary of Medicine*.

is a much more serious affection, and involves more grave consequences, both immediate and remote, than simple pericarditis.

Doctor Sibson has found endocardial complication of rheumatic pericarditis in one-half of his cases (three out of six).\* It has occurred in a proportion of somewhat more than one-third of my cases. The compound affection is characterized by the symptoms and signs of the more grave element of it; namely, the endocarditis. It is, accordingly, associated with more decided respiratory distress, and greater derangement of the circulation. Respiration is quick and panting; the pulse is very rapid, ranging usually from 120 to 140, sharp and abrupt; the heart's action tumultuous, and the cervical arteries visibly pulsating, whilst those of the limb do not seem inordinately excited.

Doctor Stokes lays much stress upon this symptom in connexion with pericarditis; and, without expressing a positive opinion, would seem to regard it, when recent, and associated with febrile action not due to inflammation or irritation of the encephalon, as diagnostic of the complication of endocarditis.† There are five different conditions, according to him, in which localized violent pulsation of the carotids may be exhibited; namely, concussion of the brain, as mentioned by Sir Astley Cooper, permanent patency of the aortic valves in the early stage, cerebritis, acute endocarditis, and exophthalmic goitre. By the absence of pyrexia, and by the positive symptoms of the disease, which cannot be mistaken, the last named affection will be readily excluded. The history of concussion of the brain, the impairment, or total loss, of consciousness, and the subsequent vertigo and vomiting, sufficiently distinguish this condition. Patency of the aortic valves will declare itself by its pathognomonic sign of basic diastolic murmur, transmitted through the aortic arch, but *not* into the arteries of the neck; and endocardial inflammation complicating pericarditis, may, though it rarely does, actually assume the form capable of giving rise to this sign; in which case carotid-pulsation would be attributable to a twofold cause; namely, one of a general, and one of a specific nature. The symptoms of cerebritis usually regarded as specific,

\* Address in Medicine, British Medical Association, August, 1870.

† *Opus citat.*, p. 52.

may be readily mistaken for the cerebral manifestations of cardiac inflammation, and the actual existence of the physical signs characteristic of the latter condition can alone be relied on for the differential diagnosis.

An endocardial murmur of a determinate character is heard within a brief period subsequent to invasion. This murmur, though in a measure masked by the more superficial, harsh, and much louder pseudo-murmur of pericarditis, may always be distinguished from the latter, firstly, by its rhythm either coinciding with, or replacing one of or both, the sounds of the heart, or immediately preceding or succeeding these sounds: in other words, by the strict association of the murmur with the sounds of the heart, as estimated by time; a character by which it is readily distinguishable from the sounds of pericarditis, which are eminently arrhythmical. Secondly, inasmuch as the endocardial murmur most often associated with pericarditis is that of mitral reflux, it must be, and actually is, audible where the pericardial friction-sound rarely reaches; viz., the left axilla and left scapular region. Thirdly, the murmur of endocarditis is at this early stage always soft and blowing in quality, unless when rendered musical, as occasionally, but very rarely, happens for a brief period, by the vibration of a flake of fibrin attached by one of its extremities to the free margin of the valve; whereas attrition-sounds are invariably rough and harsh. And, lastly, a valvular murmur, as stated by King Chambers, is perceptible to the ear raised a few lines from the stethoscope. Not so an attrition-sound.

I have never met with an example of "systolic murmur at the base of the heart from the pressure of effused lymph," as alleged by Andral,\* without, however, the production of any *post mortem* evidence in support of the assertion. No case has come under my notice in which, with false membrane engaging the roots of the great vessels, a basic murmur, when present, was not legitimately attributable to other, more generally recognized, and certainly more common, causes of this phenomenon; viz., lesion of the valves, atheroma of the root of the aorta, thrombus, or spanæmia.

\* *Anatomic Pathologique.*



Bouillaud held that *bruit de soufflet* in pericarditis occasionally depends upon thrombus in the heart, and, "conceivably," upon the pressure of liquid in the pericardium.\*

I have neither met with an example of the latter kind, nor can I conceive a degree of liquid pressure in the pericardium sufficient to resist the impetus of the column of blood escaping by the aorta or the pulmonary artery, which would not, at the same time, of necessity so impede the movements of the heart as seriously to interfere with the circulation.

Thus, it would seem, theoretically, that a murmur due to the cause alleged should be associated with partial failure of the heart and its consequences.

Hope maintained that excessive force of ventricular contraction is competent, of itself, to cause murmur.†

Law holds a similar opinion.‡

I have never heard a murmur attributable to this cause, and should feel great difficulty in admitting its occurrence.

Flint is of opinion that pericarditis is "almost always, if not invariably, associated with endocarditis,"§ from which it follows that, in his judgment, endocardial murmur is an all but invariable concomitant of pericardial *frottement*; and that, when present, it owes its origin, in the great majority of cases, to inflammation of the valves.

I cannot admit either of these propositions in the form in which they are presented. The association of endo- with pericarditis is, according to my experience, the exception; although, judged by the numerical proportion in which endocardial murmur is likewise present, it might be otherwise regarded.

Doctor Stokes states that, in his experience, the endocardial murmur is audible at a later period of the compound disease than pericardial *frottement*, and suggests the inquiry whether, in a certain proportion of these cases, the endocardial murmur may not be due to impeded action of the heart, through a close and firm adhesion of the pericardium already established.||

\* *Traité Clinique*.

† *The Diseases of the Heart*.

‡ *Dublin Journal of Medical and Chemical Science*, vol. viii., 1834.

§ *A Treatise on the Principles and Practice of Medicine*, 1868, p. 304.

|| *Opus citat.*, p. 34.

I cannot regard this hypothesis as probable, save to the extent to which it implies possible antecedent myocarditis, and consequent softening and yielding of the walls of either ventricle.

Doctor John Taylor has found valvular disease in about one-half of the cases of rheumatism examined by him; but the greater number of these examples he believes to be of old standing. He is of opinion that the frequency of acute endocarditis in rheumatism has been greatly overrated, and that it does not by much, if at all, exceed that of pericarditis.\* Of sixty-two examples of valve-disease reported by Dr. Barclay,† only twenty-three were associated with a history of acute rheumatism, and, even amongst these, in three the lesion did not appear to be of rheumatic origin. Hence, less than one-third of the cases were rheumatic, and in reference to twenty-six, rheumatism was positively denied. The distribution of these cases was as follows :

	Rheumatic.	Non-rheumatic.
Disease affecting both sets of valves (mitral and aortic) ... ..	12	10
Disease affecting mitral valve alone ... ..	7	11
Disease affecting aortic valve alone ... ..	1	5

The author's cases :

Disease affecting both sets of valves ... ..	2	1
Disease affecting mitral valve alone ... ..	2	1
Disease affecting aortic valve alone ... ..	0	0

There was another case (No. 7, the woman Nolan) in which temporary mitral systolic-murmur existed, but which I regarded as thereby excluded from the category of valvular lesions.

Where the two affections are not simultaneous in occurrence, and this is the rule, the pericarditis is usually the primary morbid element. In cases characterized by progressive engagement of the structure of the heart, or extension of inflammation by contiguity, from the investing to the lining membrane, or *vice versa*, the muscular substance of the organ must have been involved at some period of the disease. Hence the symptoms characteristic of myocarditis enter into the category of those actually

\* *Loco citat.*

† *Medico-Chirurgical Transactions*, vol. xxxv., November 11th, 1851.

present, and being attributed in all probability to the primary inflammation of the investing or of the lining membrane of the heart, as the case may be, give rise, so far, to a vitiated symptomatology. I have no doubt that in this way must be accounted for many of the symptoms erroneously attributed to simple pericarditis, or endocarditis, such as irregular or intermittent pulse, delirium, and convulsions. Of the single valvular lesions met with in association with pericarditis, mitral inadequacy is by far the most common; and, of those of compound character, mitral inadequacy, with aortic obstruction and inadequacy, holds the highest place numerically.

But the development of an endocardial-murmur in the early period of convalescence from pericarditis, is by no means proof of antecedent endocarditis, or of the actual existence of valvular lesion.

Doctor Stokes says: "The mere occurrence of murmur, even though immediately consequent on pericarditis, is not necessarily indicative of progressive valvular disease." He adds: "Thus much is certain, that the occurrence of murmur following pericarditis should not necessarily lead to the diagnosis of valvular disease in the ordinary acceptance of the term," for "it may occasionally be found that the murmur after existing for a period more or less extended, disappears, leaving the sounds of the heart in the natural condition, and the patient remains free from symptoms of valvular disease."\* He suggests, in explanation of temporary murmur in such cases, endocarditis not followed by deposit, atony of some portion of the muscular fibre, or alteration of the orifices, and consequent maladaptation of the valves by tonic spasm of the heart.

I have repeatedly witnessed examples of this kind (*e.g.*, Case 7, Mrs. Nolan), and am of opinion the murmur is caused by atony and expansion, at the acme of ventricular systole, of a portion of the wall of the left ventricle, and consequent elevation of one or both segments of the mitral valve through the connecting papillary muscles and tendinous chords. Such murmurs, when not manifestly hæmic in origin, are always mitral and systolic; they cease more promptly under tonic treatment, in-

\* *Opus citat.*, p. 2.

cluding iron, than under any other ; and rarely, under such treatment, exceed a few weeks in duration.

I cannot, therefore, agree in opinion with Dr. Latham that the cessation of an endocardial murmur denotes, in all cases, removal of newly deposited matter.\*

The symptoms characteristic of *myo-pericarditis* have been already incidentally mentioned ; they are such as might have been anticipated from the operation of any cause acting directly upon, and enfeebling the centre of the circulation.

Myocarditis, as a primary affection, is of exceedingly rare occurrence ; indeed, it may be stated that, independently of the knowledge of the affection derived from experience of it by extension from an inflamed pericardium or endocardium, nothing is positively known in regard to its symptomatology. It most frequently commences by extension of acute inflammation from the pericardium to the contiguous or outer layers of the muscular substance of the heart, and may proceed no farther, engaging only the external portion of the organ. I believe this is of common occurrence in acute pericarditis of an aggravated character, the really alarming symptoms of which, or those having reference to irregularity and threatened failure of the circulation, are attributable to inflammation of the muscular rather than of the serous structure of the heart. Thus it is that in the progress of pericarditis, the action of the heart and the pulse may become irregular or intermitting. Syncope may be threatened, or, on slight exertion, may actually occur, and the impulse of the heart, notwithstanding its tumultuous action, may be feeble, and the first sound masked or ill pronounced.

If the disease be arrested at this stage, fatty degeneration, but of a slow and progressive character, of the outer muscular substance of the heart is likely to ensue ; and thus it is, as I believe, that the greater number of such examples are produced. The inflammatory process extending inwards may engage the entire substance of the heart, and, if not immediately fatal, may give rise either to suppuration, or to fatty degeneration of its structure, according to the activity of the preceding inflammation. The early stage of inflammation of the heart, or that of acute conges-

\* *Lectures*, vol. i., p. 149.



tion, is characterized anatomically by dark red discoloration of the portion of its substance engaged; the muscular structure, so altered in colour, is soft, and easily torn. After a period of variable length, it becomes light in colour, or assumes a yellow tint, according to the rapidity of the disorganizing process. If suppuration set in, the muscular substance becomes infiltrated with pus, light in colour, and lacerable in the highest degree; minute abscesses may be formed in various portions of its substance, and may lead to ulceration.

The process of fatty transformation of the muscular substance of the heart is much less rapid, and the result of more chronic inflammatory action.

Fatty metamorphosis of the heart may owe its origin to endocarditis, and commence at the internal surface of one or more of the chambers, thence slowly progressing outwards, and ending either in suppuration or in fatty degeneration, to a greater or less extent, of the substance of the heart. The characteristic features of such a case would be those of endocarditis, modified by symptoms of partial and momentary failure of the heart. Thus, there would be precordial oppression, quick and tumultuous action of the heart, and hurried respiration. To these would succeed quickly, it might be within a few hours, irregularity and intermission of the heart and pulse, vertigo, and temporary loss of consciousness, paroxysmal dyspncea, and collapse of the features.

From this alarming condition partial restoration may be effected, to the extent of enabling the patient to resume his ordinary duties; but, fatty transformation of the substance of the heart, whether immediate or consecutive to purulent infiltration, once started, is progressive, and leads inevitably to death at a more or less remote period, according to circumstances influencing the rate of its advance.

Those who are obliged to labour bodily or mentally to obtain a livelihood, more readily succumb to this process, owing to the twofold cause of rapid molecular disintegration, and imperfect nutrition of the heart. Those who, on the contrary, are in a condition of life which enables them to have repose, and at the same time suitable nutriment, may live to a comparatively advanced

age, even though they be actually the subjects of fatty transformation of the heart commencing in endo- or pericarditis.

The triple combination implied by the compound title *endomyo-pericarditis* is of comparatively rare occurrence. When met with, it is characterized by a corresponding combination of signs and symptoms; viz., the valvular murmur of endocarditis, the friction-sound of pericarditis, and the symptoms of failure of the heart, already detailed, by which myocarditis may be confidently diagnosed. Amongst the latter symptoms, those having reference to the cerebrum, and presented under the forms of delirium, convulsions, and coma, may be appropriately discussed here.

*Delirium* is mentioned by Corvisart\* and Bertin† as a symptom of acute pericarditis, without, however, any statement of opinion as to its cause. Bouillaud likewise mentions, as a symptom of pericarditis, "delirium, usually slight and momentary, sometimes violent," and occasionally followed by convulsions.‡

Andral gives the particulars of a case of pericarditis in which delirium was a prominent symptom. A young woman, after a brief illness, became delirious; there was improvement and return of consciousness, twitching of the muscles of the face, occasional tetanic spasm of the upper extremities, return of delirium, coma, and death. Soft exudation was found on the pericardium, with some bridges of lymph extending from surface to surface, and, in the sac, some ounces of green flocculent serum.§

In this case the effusion present in such small quantity could not have been the cause of the cerebral symptoms; whilst its colour and quality indicated that the substance of the heart was involved in the inflammatory process, although, in appearance, it is described as "presenting nothing morbid." He quotes a similar case from Rostan|| and attributes the head symptoms, in both cases, to a "sympathetic affection of the brain," by which

\* *On Diseases of the Heart*, Hebb's translation.

† *Traité des Maladies du Cœur*, 1824, p. 252.

‡ *Traité Clinique des Maladies du Cœur*, 1835.

§ *Clinique Médicale*, vol. i., second edition, 1829, observation viii.

|| *Recherches sur le Ramollissement du Cerveau*, p. 233.

he manifestly means to convey failure of the circulation in that organ.

Doctor Latham mentions two cases of pericarditis in which the cerebral symptoms were so pronounced as to resemble those of inflammation of the brain,\* and Hope includes delirium and convulsions amongst the symptoms occasionally witnessed "in the last stage," and regards them as the result of "cerebral congestion, and the circulation of venous blood;"† in other words, as symptoms of asphyxia due to slow failure of the heart and congestion of its right cavities. Dr. Sibson details the case of a man, aged twenty-eight, a free liver, who, on the evening of the sixth day of acute rheumatic pericarditis, became violently delirious, and presented symptoms generally resembling those of delirium tremens. He died on the eighth day. Both layers of the pericardium were found covered with honeycombed lymph. No mention is made of the condition of the heart substance.‡

Sir T. Watson gives the particulars of four cases of rheumatic arthritis, in which the heart was affected and symptoms suggestive of cerebral inflammation also existed. The only pathological changes, however, exhibited by the brain in these cases, were slight serous infiltration of the pia mater, and partial effusion into the lateral ventricles.§ Dr. Bright details the case of a young man who, on the sixth day of an attack of acute rheumatism, became excited and delirious; the symptoms became more urgent, and it was found necessary to put the patient under restraint. At the end of three weeks he died. The brain was found perfectly healthy, but the pericardium and the endocardium exhibited evidence of recent and acute inflammation.|| Dr. G. Burrows records an example of death in a state of delirium after seven days' illness. The brain was, to all appearance, unaltered, but the pericardium was covered with recent lymph, and, over a small space, with concrete pus.¶ Dr. Todd furnishes a no less striking example of a similar kind. A young woman,

\* *London Medical Gazette*, vol. iii, p. 209.

† *Opus citat.*, p. 156.

‡ *Lectures on the Principles and Practice of Physic*, fourth edition, vol. ii., p. 303.

§ Address in Medicine, British Medical Association, 1870.

|| *Medico-Chirurgical Transactions*, vol. xxii.

¶ *Disorders of the Cerebral Circulation*.

some days ill of rheumatic fever, was seized with delirium, which was succeeded by convulsions, coma, and death. The brain and its membranes exhibited no inflammatory product, and were even paler than normal, whilst the pericardium was covered with recently exuded lymph.\* Dr. Fuller is of opinion that the cerebral symptoms exhibited in the course of acute rheumatism, are due to the toxic action of the rheumatic virus on the brain.† Trousseau regards cerebral manifestations in acute rheumatism as the result of a "neurosis," and maintains that they may be primary; preceding, or even replacing the rheumatic affection of the joints or of the heart, and meriting the designation of "cerebral rheumatism,"‡ according to him. Todd§ and Fuller|| are of opinion that an organic poison, the product of a morbid process associated with rheumatism, constitutes one and the principal factor in the production of the cerebral phenomena; but that another and no less important factor is a morbid impressionability of the cerebro-spinal nerve centres, hereditary or acquired.

The presence, within the cranium, of the pathological products of acute inflammation; viz., lymph and pus, has been observed by Watson and Fuller. The association, however, is exceedingly rare, and may have been due to a simple coincidence. In acute cerebro-spinal meningitis, the occurrence of articular pain and tumefaction is not uncommon; but, the order of manifestation, and the general character of the symptoms of this disease, would preclude the possibility of confounding it with articular rheumatism.

Da Costa says that in pericarditis the thoracic symptoms may be in abeyance, and others, indicative of gastric or cerebral irritation or inflammation, may take their place; these latter being, in his opinion, often due to the poison of rheumatism, or to that of Bright's disease.¶

Flint mentions, as occasionally witnessed amongst the symp-

\* *Lectures on Delirium and Coma.*

† *On Rheumatism and Rheumatic Gout.*

‡ *Clinical Medicine*, vol. i., p. 534, Sydenham Society's edition.

§ *Lumleian Lectures, Lancet*, 1852.

|| *Loco citat.*

¶ *Medical Diagnosis*, third edition, 1870, p. 358.



toms of pericarditis, paroxysms of maniacal excitement, alternating with alarming dreams, and starting from slumber.\*

Doctor King Chambers thinks that delirium in rheumatism indicates vascular and nervous depression, requiring stimulants, tonics, and opium; and that it is more frequently symptomatic of pericarditis or pneumonia than not.†

The different opinions held as to the pathogenesis of cerebral symptoms, associated with rheumatic inflammation of the heart or its membranes, may be conveniently presented in synoptical form.

Partial failure or derangement of cerebral circulation, through inflammation and consequent debility of the heart	...	<div> Andral.  Watson,  Burrows. </div>
Rheumatic poison acting on susceptible nerve-centres	...	<div> Trousseau.  Todd.  Fuller.  Da Costa. </div>
Cerebral congestion and circulation of venous blood	...	Hope.
Vascular and nervous depression (dynamic ?)	...	King Chambers.

It would appear, therefore, that pericarditic delirium and convulsions have been attributed by writers to two different and nearly opposite pathological conditions; namely, defect of arterial circulation in the brain through failure of the left heart, and the circulation through that organ of a blood vitiated by an organic poison elaborated within the body itself, or by retained excreta.

I have not witnessed well pronounced delirium or convulsions in connexion with pericarditis. Occasional wandering of mind, and partial incoherence, easily dissipated by sharply addressing the patient, and such as is commonly witnessed in essential or inflammatory fever, have frequently come under my notice in the first and second stages of pericarditis. In the stage of serous effusion, accompanied with engorgement of the cervical veins, lividity of the features, and failure of the heart, I have likewise witnessed incoherence. Mental obscuration is usually manifested in partial asphyxia from copious effusion, under the

\* *A Treatise on the Principles and Practice of Medicine*, Philadelphia, 1868, p. 304, *et sequent.*

† *Lectures, chiefly Clinical*, third edition, 1864, p. 142.

form of somnolence and muttering, contraction of the pupils, and slowness in collecting the thoughts; on being roused to the effort, however, the patient is capable, after a little hesitation, of giving a connected answer.

In the former case, the slight mental derangement present is due, as in ordinary febrile excitement, to rapid disintegration of tissue and imperfect excretion; in the latter it is the consequence of failure of the *right* side of the heart, and venous engorgement of the brain.

*Syncope* has been noticed in connexion with pericarditis by Bertin, who witnessed it in two cases out of thirty-six,\* and mentions faintness, failure of the pulse, and anxiety, as amongst the ordinary symptoms.†

Louis had not an example of syncope amongst his four cases, and he denied that it was an essential symptom of pericarditis.‡ Neither have I witnessed an example, unless fatal syncope, by which several of the cases that had proceeded to the stage of liquid effusion terminated, be admitted into this category.

*Traumatic pericarditis* presents no peculiar features by which it is distinguishable from the ordinary or idiopathic form of the affection, save those imparted to it by the general and local effects of the mechanical lesion. Dr. Stokes gives the particulars of a case of this kind resulting from a gun-shot wound, a charge of small shot having been lodged in the superficial structures of the precordium; after the lapse of a few days the symptoms and signs of pericarditis were exhibited, the latter being characterized by a number of distinct seats of friction-sound of various intensity and duration. The patient recovered, and no opportunity was therefore presented for determining the precise depth or character of the injury; but, it is presumed that a few pellets had actually penetrated the pericardium, and been so many independent centres of inflammation.

Flint records a very remarkable example of the same kind. A person in a state of intoxication swallowed a set of false teeth, which lodged in the œsophagus, and effected a communication

\* *Traité des Maladies du Cœur*, 1824, observation lxiii., p. 239.

† *Opus citat.*, p. 270.

‡ *Mémoires ou Recherches Anatomico-Pathologiques*, 1826.

with the pericardium by ulceration. The specimen is preserved in the Bellevue Hospital, New York.\*

Amongst the *intrinsic complications* of pericarditis are included tubercular deposition in, and cancerous and calcareous transformation of, the pericardium. Of the two former, mention is made by Bonetus† and Hasse;‡ of the latter, examples are quite numerous, and several have been already adverted to. The process of deposition, or of tissue-transformation, would seem in every case to engage the false membrane or neoplastic structure only, at least in the first instance.

The *extrinsic complications* of pericarditis may be classified under two heads, constitutional and local. Those of a general or *constitutional* character most frequently met with are Bright's disease in its various forms; the exanthematous fevers, especially scarlatina; diffuse inflammation; pyæmia; gout; and, preeminently, rheumatism.§

Doctor John Taylor states that of thirty-five cases of aggravated pericarditis, one-third nearly were associated with Bright's disease, and two-thirds with acute rheumatism. He has found acute pericarditis as a secondary inflammation in the advanced stages of Bright's disease, in the proportion of one in ten.||

Doctor Mayne returns five out of his eleven cases of pericarditis as rheumatic.¶ Eight of my cases, thirteen in number, were of rheumatic origin. Out of a total of seventy-four cases of acute and subacute rheumatism reported by Dr. Sibson, six only were affected with pericarditis; and of twenty-three cases of

\* *A Treatise on the Principles and Practice of Medicine*, 1868.

† *Sepulchretum*.

‡ *Pathological Anatomy*, Sydenham Society, 1846, p. 116.

§ In the strict sense, the term "complication" is not applicable to the above-mentioned diseases in their relationship to pericarditis; they constitute, in each instance, rather the original disease. To an author, however, treating of pericarditis as a distinct and independent entity, it is convenient, even though its secondary importance, pathologically and therapeutically, in any given category, be admitted, to regard concurrent or coexistent disease, of whatever kind, as a complication. Indeed, in regard to any disease under special consideration, all other diseases having any relationship to it, might be conveniently classified under the three heads of *causal*, *casual*, and *consecutive* complications. In this sense the diseases mentioned in the text would belong to the first of these three classes.

|| *London Medical Gazette*, new series, vol. i., 1845.

¶ *Dublin Journal of Medical and Chemical Science*, vol. vii., 1835.

acute gout returned by the same author, only one was affected with pericarditis; and even this one was of a transient character.\* I have already adverted to the opinion of Trousseau, to the effect that the pericarditis of scarlatina is likewise of rheumatic origin, being due to a rheumatic complication of that disease frequently witnessed under the form of arthritis, rather than to the poison of scarlatina.† This is a very questionable doctrine, even to the extent of assuming the existence of the twofold poison. It is, at best, only an hypothesis, which is not susceptible of proof or open to refutation, and manifestly not necessary to a full explanation of the facts. Intercurrent inflammation and swelling of various structures and organs are common in scarlatina; and serous membranes, other than the pericardium, are frequently implicated. Dr. Law remarks, with truth, that where pericarditis arises from, or in connexion with, diffuse inflammation, the effusion is likely to be purulent.‡

Amongst the extrinsic *local* complications of pericarditis may be mentioned pleuritis, pneumonia, pleuro-pneumonia, and hepatitis. In most of the cases of pericarditis published by Corvisart, one or other of these complications was present,§ most frequently pleuro-pneumonia and diaphragmatic pleurisy. Gastritis has been likewise mentioned by Corvisart as a frequent complication; but, manifestly, on insufficient evidence, as the only symptom pointing to gastritis indicated by him was epigastric tenderness, a symptom now generally attributed to pericarditis itself, but in many instances due to hepatic congestion.

Inflammation of the lungs accompanied pericarditis in more than one-half of Dr. Latham's cases, and endo-pericarditis in more than two-thirds; whilst of endocarditis alone the proportion of cases in which pulmonary inflammation existed was only one in nine.||

Graves details an example of pericarditis complicated with, and dependent upon, hepatic abscess, as already quoted.¶

\* Address in Medicine, British Medical Association, 1870, pamphlet, p. 24.

† *Clinical Medicine*, vol. i., p. 397.

‡ *Reports of Pathological Society of Dublin*, vol. i., p. 42

§ Hebb's translation, p. 6, *et sequent.*

|| *London Medical Gazette*, new series, vol. i., 1845.

¶ *Clinical Medicine*, vol. ii., p. 233.



Doctor John Taylor gives an example of pericarditis by extension of inflammation from the pleura in one instance, and from the liver and diaphragm in another.\*

Acute pericarditis is a disease of *early* and *middle* life. I have repeatedly seen it in early childhood, but never in persons over forty years.

Louis mentions a fatal case of pericarditis in a child of seven months, which had been related to him by Boudard,† and is of opinion that the periods of life at which pericarditis is most common, are between seventeen and thirty, and between fifty and seventy years, respectively. I have never seen a case of pericarditis within the latter period, except in the chronic form, and as a complication of advanced renal disease.

Out of fifty-five cases of pericarditis given by Hache, only six were over forty years of age; and Fuller declares that pericarditis is of much more frequent occurrence in youth than in advanced age.‡

Chronic pericarditis, on the contrary, is most common in the aged; because this is the period of life in which chronic visceral disease, of which it is usually a complication, especially renal disorganization, is most prevalent.

In regard to the relative proportion of the *sexes* affected with pericarditis, most authors agree that the majority are of the male sex.

Out of one hundred and seven cases given by Louis,§ only twenty-seven were females; and in those published by Bizot, seven in number, one only was of that sex.||

Bouillaud reports only six females out of a total of thirty-four cases of pericarditis.¶

Hache estimates the proportion as one female to four males.\*\*

Of thirteen cases given by the author in the preceding table (p. 360), nine belonged to the male sex.

\* *Loco citat.*

† *Mémoires ou Recherches Anat. Pathol.*, p. 292.

‡ *Archives Générales de Médecine*, tom. ix., November 1835, p. 328.

§ *Diseases of the Chest*, p. 513.

|| *Loco citat.*

¶ *Traité Clinique des Maladies du Cœur.*

\*\* *Loco citat.*, p. 119.

Sibson's six cases of pericarditis were in males, and all under thirty-four years of age.\*

The preponderance of males amongst those affected with pericarditis may, therefore, be regarded as established. This will probably be satisfactorily explained by reference to the more laborious occupations and greater exposure to vicissitudes of weather and changes of temperature, of males of the working classes, who are most frequently the subjects of pericarditis, whether rheumatic or renal.

Fuller maintains that females are more liable to rheumatic pericarditis than males,† and Dr. Barclay declares that this is likewise true of other cardiac complications of rheumatism.‡

There is no divergence of opinion, however, between these writers and those previously quoted, because they allege only what is admitted by all; namely, a larger per centage of pericarditis, relatively to the number affected with rheumatism, amongst females than amongst males.

Rheumatic pericarditis is fatal in the proportion of about one in six cases, according to Fuller§ and Louis. I have had only one death in eight cases (see table, p. 360).

The *prognosis* in pericarditis should be determined rather by the complications than by the disease itself. Simple pericarditis is rarely fatal. In patients of strumous cachexia, however, serous effusion into the pericardium, causing death by paralysis of the heart, has occasionally taken place. The opinion of Corvisart, to the effect that acute and chronic pericarditis are always fatal, the subacute form being only a degree less unfavourable, will now find few if any subscribers, and is explicable only by the imperfect state of the science of diagnosis at the time he wrote. It will be remembered that at the date in question the principles of mediate auscultation were still unknown, and that, consequently, those cases of pericarditis which were of a sufficiently formidable character to make themselves manifest by *symptoms* so pronounced as not to admit of misinterpretation,

\* *Loco citat.*

† *Opus citat.*

‡ *Medico-Chirurgical Transactions*, vol. xxxv.

§ *Opus citat.*

were alone diagnosed. The diagnosis of pericarditis, in the acute form, was based upon the general effects of liquid effusion into the pericardium; and, in the chronic form, upon the symptoms indicative of adhesion of the pericardium with hypertrophy and dilatation of the heart. That Andral laboured under the same disadvantage may be inferred from the following declaration: "And if no pain exist in the region of the heart, there only remains to recognize the pericarditis, the dull sound of this region."\*

Flint truly remarks that the prognosis depends upon the complications; and that it is most unfavourable in Bright's disease, and in cases of copious effusion rapidly poured out. In regard to the issue of pericarditis in its milder form, I unreservedly subscribe the following rule of practice laid down by Dr. Stokes: "While any signs continue, he (the patient) must be carefully watched; for, in certain cases, a sudden change occurs, and the disease is converted from an apparently trivial and latent affection into a more severe form."† The prognosis is less favourable in those of weak constitution than in the strong, and in childhood than in adult age, for the reason previously stated.

It is supposed by some writers that the *season* exercises a definite and perceptible influence on the prevalence of pericarditis. Thus, Hache declares that nearly one-half (eighteen out of thirty-eight) of the cases recorded by him occurred in the last four months of the year, and only one-fourth in the first four; viz., from January to May.‡ Inasmuch as the prevalence of pericarditis is mainly regulated by that of acute rheumatism, of which it is usually a complication, the season and the climate most favourable to the latter will obviously be those in which pericarditis will be most prevalent.

Doctor Fuller shows that within the first four months of the year, rheumatism is slightly more common than during the corresponding periods of the middle and terminal portions.§

I have found rheumatism more prevalent during the cold fogs

\* *Clinique Médicale*, vol. i., p. 38, foot note.

† *Opus citat.*, p. 45.

‡ *Loco citat.*

§ *Rheumatism, Rheumatic Gout, and Sciatica*, third edition, 1860, table iv., p. 44.

of November and December than at other periods of the year; and, in like manner, its most frequent complication, pericarditis. In all countries and seasons a moist atmosphere has been found favourable to the production of rheumatism and pericarditis.

The *treatment* of pericarditis resolves itself into that of the disease in its acute, chronic, and recurrent forms respectively.

At the outset of acute pericarditis in the young and vigorous, Bouillaud and Hope urge the abstraction of blood, *coup sur coup*, by means of the lancet, and, locally, by leeches or the cupping-glass. A daily bleeding from the arm of twenty to thirty ounces, followed by an equal loss locally, for two or three consecutive days, would not have been considered by them as an excessive amount in the treatment of the first stage of acute pericarditis, in a young subject of previously unimpaired health.

I have never had to treat a case of pericarditis that seemed to require, or even to warrant, so vigorous a plan of treatment. I prefer, in every case, local abstraction of blood by means of leeches; ten, twenty, or thirty, according to the urgency of the case, and the strength and constitutional tolerance of the patient. This measure might, if necessary, be repeated once or oftener, and should be invariably succeeded by the application over the leech-bites of a warm poultice. The necessity for a repetition of leeching should be determined by the impulse, rhythm, and sounds of the heart, and by the sensations of the patient as referred to the heart. If the impulse be strong and abrupt, the rhythm regular, the sounds, especially the first, normal or sharp, and a sensation of oppression or pain at the precordium be still experienced by the patient, leeching should be repeated to such extent as may be deemed necessary to effect the object held in view; namely, to arrest the progress of inflammation and subdue these urgent symptoms. I have never used the cupping-glass for this purpose in the first instance, as I should apprehend the possible occurrence of unpleasant consequences, such as syncope, from the shock of its application. I can, however, see no objection to the use of a dry cup for the purpose of abstracting blood from lancet-punctures or leech-bites. Immediately blood in sufficient quantity has been abstracted by a first leeching, my invariable practice in acute cases is to administer mercury in



small and frequently repeated doses. I usually direct that half a grain of colomel, with one grain of James' powder, shall be given in powder or pill every hour. For the James' I substitute Dover's powder if there be a tendency to diarrhœa; and if it actually exist I give grey powder or blue pill with opium, in equivalent doses at corresponding intervals. I have found this mode of administering mercury, commonly known as that of Dr. Law, in acute sthenic inflammations, more rapidly efficacious, and much more manageable, than that of giving it in doses of ten to twenty grains twice or thrice daily, as proposed and practised by Dr. Johnson and sanctioned by Dr. Graves. Administered in this manner, I push the use of mercury to slight salivation, and am satisfied when the imprint of the teeth, with white and elevated lines of union, appear upon the inner surface of the cheeks; when the gums present a swollen, soft, and spongy appearance, and when coppery taste is experienced, and a slight drivelling of saliva at the angles of the mouth takes place. These results are usually attained, in cases of ordinary impressionability, within a period of twenty-four hours, and simultaneously a noticeable improvement in the patient's general condition takes place; the pulse has descended in frequency, and become relaxed, the surface temperature reduced, and, perhaps, the skin moist, breathing less rapid and embarrassed, and the feeling of oppression at the heart less urgent, or entirely abolished.

A saline aperient should be administered at the outset, if the bowels be not actually over relaxed. I usually prescribe a seidlitz powder with a drachm of syrup of ginger; or two drachms of the acid tartrate of potash, with a drachm of tincture of ginger, and two ounces of the infusion of senna.

When the symptoms of improvement above described have appeared, under the treatment just sketched, the use of wine, in moderate doses, and frequently repeated, should be immediately commenced, with a view to obviate such a state of cardiac debility and relaxation of the coronary vessels as might favour the occurrence of serous effusion into the pericardium. It should be given in doses of at least one ounce every second hour; and, above this minimum, the quantity should be regulated by the impulse and sounds of the heart, and the quality of the

radial pulse. If the cardiac impulse be of moderate force, the first sound distinct and clear, and the second sound single, the pulse at the wrist being full and monocrotic, the minimum allowance of wine may be deemed sufficient. But if, on the contrary, the impulse of the heart be feeble, the first sound muffled and indistinct, and it may be, the second sound double, whilst the pulsations of the radial artery are small, feeble, or dicrotic, and perhaps occasionally lost, wine in much larger doses, and given every hour, would be demanded. To those who have been addicted to the free use of ardent spirits, I allow, in preference, whiskey or brandy in equivalent quantity.

Whilst treating of this subject, Dr. Stokes remarks: "In truth, it may be said, that no man is fit to treat general disease, or local inflammation, especially its secondary forms, until he has conquered that fear of stimulants, which a long course of erroneous teaching has instilled into his mind."\*

Doctor Todd has been charged, or rather his memory, with having, in his posthumous work edited by Dr. Beale,† advocated the use of alcoholic stimulants in the treatment of acute disease to an unwarrantable extent, and set thereby a dangerous example to less judicious practitioners. I can only say that, having read this lecture with the greatest care and attention of which I am capable, I failed to discover in it any justification for this charge. Dr. Todd advocates the *rational* use of stimulants, somewhat as I have above recommended them; and after a full and able exposition of their physiological action, the circumstances under which they are admissible, and the benefits derivable from their use, he says, "Alcohol is a form of aliment appropriate to the direct nourishment of the nervous system, and to its preservation. Taken carefully, it increases the animal temperature; it also strengthens the action of the heart; and when administered under proper circumstances it reduces the frequency of the pulse.

"You must give enough to keep up animal heat, and to protect the tissues without embarrassing the stomach, and without allowing the fumes of alcohol to be perceived in the breath.

"Alcohol may be employed in all those diseases in which a

\* *Diseases of the Heart and Aorta*, p. 90.    † *Lectures*, 1861, lecture xvi.

*tendency* to depression of the vital power exists, and there are no acute diseases in which this lowering tendency is not present.

“Delirium is a symptom of enfeebled and contaminated nutrition of the brain. It is to be looked for in all exhausting diseases, and in all acute maladies accompanied by high fever.

“When alcohol is given, as I have recommended, it calms the nervous system, promotes tranquil sleep, from which the patient may be easily roused, and averts delirium.

“The rule of its administration should be ‘a little and often,’ from two to sixteen drachms, more or less dilute with water or other bland fluid, and repeated at such intervals as his state of vital power may seem to demand.”

From the preceding extracts, it will be manifest that Dr. Todd was by no means an advocate for the free and indiscriminate use of alcohol in acute disease, as has been alleged. On the contrary, the indications for its use, and the rules for its administration both as to quantity and mode, which he lays down, are those which guide the most enlightened practice of the present time.

In regard to the effect of alcohol upon the temperature of the body, however, exact experiment has recently established, contrary to the inferences of Dr. Todd, that it is either neutral, or positively depressing, according to the actual state of health of the subject of experiment. Thus, Dr. Parkes has shown that it in no degree affects the temperature of a healthy body, and does not increase the body weight, or affect the elimination of nitrogen by the bowels or kidneys; whilst Professor Binz has experimentally proved that alcohol sensibly and continuously lowers temperature in fever.

Clinical experiment and observation have decisively sanctioned the enlightened use of alcoholic stimulants in most acute diseases. But, in dealing with those acute inflammatory affections in which the mass of the blood is brought into immediate contact with the inflamed surface, as in endocarditis, endarteritis, and phlebitis, alcoholic agents, though occasionally demanded, should, as a rule, be eschewed.

An opiate sedative, consisting of a grain of the watery extract, or fifteen minims of Batly's sedative, should be given at

night, with the view of insuring sound sleep, by which the excitement of the heart is allayed. I find opium, in some one of its forms, the most suitable and efficacious hypnotic in such cases; it is usually well borne, even in large doses.

The diet should be bland and nutritious, consisting of beef-tea, chicken-broth, arrowroot, etc.; and, after acute febrile action has subsided, solid food in small quantity, such as a chop, or the breast of a chicken, may be allowed, provided the tongue be moist. For drink, saline beverages should be given, such, for example, as the so-called "imperial," consisting of an ounce of bitartrate of potash dissolved in a quart of water, flavoured with lemon-juice, and sweetened to the taste; milk and soda water in equal proportions; or the natural alkaline waters of Vichy, Vals, Ems, or Apollinaris.

When the acute inflammation has been subdued, as indicated by subsidence of acute febrile symptoms, I substitute for the previous lowering treatment some tonic medicine, such as decoction of cinchona with iodide of potassium, in doses of three grains of the latter to an ounce of the former; or infusion of gentian with bicarbonate of soda; and, in every case, wine or whiskey at this stage, in quantity determined by the general condition and previous habits of the patient.

Doctor Stokes sanctions a single bleeding from the arm in a case of uncomplicated pericarditis in the earlier stages, but prefers abstracting blood locally by means of relays of leeches, from ten to twenty, repeated three or four times within the twenty-four hours if necessary, and succeeded by warm poultices. He advocates the administration of calomel at the same time, and recommends that it shall be given in large doses after the method of Graves.\* The use of calomel with opium is recommended also by Fuller,† and, indeed, by most modern authors.

Opium has been freely administered in acute pericarditis, with a view to allay the excitement of the heart, at the same time that topical abstraction of blood is being effected. This practice, which certainly has its advantages in the treatment of acute pericarditis associated with great nervous excitement, pal-

\* *Opus citat.*, p. 84.

† *Rheumatism, Rheumatic Gout, and Sciatica*, third edition, 1860.



pitiation, and feeble circulation, as in anæmic and hysterical females, has been lately advocated very strongly by Dr. Frederick J. Farre,\* who reports most favourably of it. He gives eleven cases of cure, under a plan of treatment consisting in the administration of one grain of opium every sixth hour.

Doctor Chambers, likewise, strongly recommends opium in the treatment of both acute rheumatism and rheumatic pericarditis.† He no less decidedly disapproves the use of mercury in the treatment of the former, on the ground that, by increasing relatively the proportion of fibrin in the blood, it predisposes to pericarditis.

In the second stage of acute pericarditis, after the subsidence of febrile action, I would, in addition to the treatment above indicated, namely, iodide of potassium and alkalized bitters, make use of counter-irritation in some form, with a view to keeping up a derivative action from the heart; and with the same object in view, I would reapply leeches at any period of this stage, should the evidence of renewed inflammation, especially a return of precordial pain, be presented. Probably the best form of counter-irritant in such a case would be the vesicating collodion, or an ordinary blister brushed over with this fluid, the object being to effect rapid as well as complete vesication. A warm poultice continuously applied over the blister will accelerate its action, but its weight cannot, in many cases, be borne. As a substitute for a blister, I have frequently used in this stage a mercurial and opiate plaster, consisting of half an ounce of the ordinary mercurial plaster of the Pharmacopœia, with ten grains of opium, spread upon strong paper and laid over the heart, as first recommended by Dr. Lionel Beale. I give a preference to this application, when much nervous excitement of the heart exists, with loud and grating friction-sound.

The use of digitalis may be demanded at this period of the disease. If tumultuous, and at the same time irregular action of the heart occur, digitalis, in doses of five to ten minims of the tincture, given every third hour, will be found efficacious in lowering the rate, restoring the rhythm, and increasing the

\* *St. Bartholomew's Hospital Reports*, vol. ii.

† *Lectures chiefly Clinical*, 1864, p. 126.

strength, of the heart's action.\* Colchicum and bark have been recommended in this stage; but, Stokes truly remarks that these medicinal agents should be given with caution where fever or cardiac excitement exist.

In a comparatively small proportion of cases, pain of a paroxysmal character, and resembling that of angina pectoris, occurs. In such cases it will probably be found that the other symptoms and signs lead to the diagnosis of myo-pericarditis. In the treatment of this affection, Dr. Latham has found opium in comparatively large doses to act most beneficially as an anodyne and sedative. In combination with chlorodyne I have found it of the utmost value. Ten to fifteen minims of chlorodyne with an equal quantity of Battley's sedative, given in an ounce of water, sufficing usually to insure relief from urgent pain and cardiac distress.

In the third stage I rarely give digitalis, because of the existence of a mechanical impediment to the movements of the heart; the exceptions are cases in which, other measures having failed to dissipate a serous accumulation in the pericardium, I would try its effects as a diuretic, before having recourse to the *dernier ressort*, paracentesis.

In the treatment of liquid collections in the pericardium I have no faith in blisters, and rely mainly upon diuretics and hydragogue purgatives. Of medicines of the former class, acetate of potash in half drachm doses, given, every second hour, with a drachm of spirits of juniper in an ounce of compound decoction of scoparium, is that to which I usually give a preference; and as a purgative, I give the compound powder of jalap in doses of half a drachm to a drachm, with two to four grains of calomel, according to the age, strength, and susceptibility of the patient. The allowance of liquids should be, at the same time, restricted to the smallest possible amount; indeed, the patient should be urged to allay rather than satisfy thirst, by frequently rinsing the mouth, or sucking a morsel of ice. During the entire of this period wine should be given, in quantity determined by the force and quality of the pulse.

\* I would not venture the application of powder of digitalis to a blistered surface over the heart, as recommended by Bouillaud.

In many cases, the best directed and the most active medicinal measures will fail to dissipate serous collections in the pericardium. In such cases, the sac is usually distended, the heart is impeded in its movements, the pulse is irregular and failing, and the patient's life is in imminent jeopardy. At any moment, under these circumstances, death may suddenly occur by syncope on the patient's making the slightest muscular effort, such as that involved in sitting up to drink, or even turning in bed. One alternative alone now remains to the physician, and one which, if it is to save life, must be promptly put in execution; namely, *paracentesis pericardii*.

Senac advocated this operation in the treatment of hydro-pericardium, and recommended, as the best instrument for the performance of it, the trocar and cannula. He has not, however, adduced even a single case in his own practice to exemplify the benefit derivable from the operation.

Corvisart believed "that the advantage to be derived from this operation will rarely counterbalance the danger which attends it;"\* but should it be decided upon, he gives a preference to the bistoury for its performance.

Desault actually essayed the operation; but, having proceeded upon a wrong diagnosis into which he and his colleagues were led, namely, that of mistaking a circumscribed pleuritic effusion for hydro-pericardium, he did not enter the pericardium, as was proved by examination of the body after death.

Doctor F. A. Aran has successfully performed the operation, and subsequently injected iodine into the pericardium: the patient recovered.† The solution injected consisted of equal proportions of tincture of iodine and water, with one-fourth part, by weight, of iodide of potassium.

Successful performance of the operation, followed by recovery of the patient, has been likewise recorded in the practice of Jobert de Lamballe,‡ Romero of Barcelona (two examples),§

\* *Opus citat.*, p. 52.

† *Bulletin de l'Académie de Médecine*, tom. xxi, p. 142; and *Dublin Hospital Gazette*, December 1st, 1855.

‡ *Trousseau's Clinical Medicine*, Sydenham Society's edition, vol. iii., p. 371.

§ *Dictionnaire des Sciences Médicales*, vol. xl., 1819.

Wheelhouse,\* and Karawagen.† The procedure has been practised, successfully as to operation but unsuccessfully in regard to result, by Trousseau, Larrey, Schuh, Heger, Romero, Wheelhouse, and Karawagen, as exemplified in the following table:

TABLE IV.

Operator.	Successful.	Unsuccessful.	Erroneous Diagnosis.	Mode and Site of Operation.	Death.	Recovery.
Desault ... ..	—	1	1	Bistoury, between fifth and sixth ribs	1	—
Trousseau ... ..	1	—	—	Bistoury, below nipple	1	—
Jobert de Lamballe	1	—	—	Bistoury and trocar in fifth intercostal space	—	1
Larrey ... ..	—	1	1*	Ditto	1	—
Schuh ... ..	1	—	—	Trocar and cannula, between third and fourth ribs, near sternum	1	—
Heger ... ..	1	—	—	Trocar and cannula; two operations	1	—
Romero ... ..	1	—	—	Bistoury and scissors, between fifth and sixth ribs	—	1
Romero ... ..	1	—	—	Ditto	—	1
Romero ... ..	1	—	—	Ditto	1	—
Bowditch ... ..	—	—	—	Bistoury	1	—
Roux ... ..	—	1	1			
Aran ... ..	1	—	—			
Aran ... ..	1	—	—	Bistoury and capillary trocar, in fifth intercostal space	—	1
Aran ... ..	1	—	—	Ditto	—	1
Wheelhouse ... ..	—	—	—	Ditto	—	1
Wheelhouse ... ..	1	—	—	Capillary trocar, in fifth intercostal space	1	—
Karawagen ... ..	1	—	—	Not stated	—	1
Karawagen ... ..	1	—	—	Ditto	1	—
18	13	3	3		9	7

\* Uncertain. No P. M.

\* *British Medical Journal*, 1868.

† *British and Foreign Medico-Chirurgical Review*, vol. xii., July, 1841.



Thus, out of a total of eighteen cases, thirteen were successful in regard to the immediate object sought; namely, the removal of liquid from the pericardium; three were in this respect unsuccessful, and in each instance failure was due to an error of diagnosis; death followed in nine instances, and recovery in seven. In eleven instances the bistoury was the principal instrument used, and in three, the trocar and cannula. In no instance, to my knowledge, has the pneumatic aspirator been used in the performance of this operation.

I have never yet performed the operation, nor had it done by another; but, on two occasions, at least, I have had reason to regret the omission of its performance. The circumstances which warrant, and even demand, the operation, as it seems to me, are the following.

If the presence of copious liquid effusion in the pericardium be indubitable, as determined by the evidence previously insisted upon (p. 387), and there exist, at the same time, urgent dyspnœa, small and failing pulse, œdema of the lower limbs and face, and venous engorgement of the neck, medicinal agency having failed to disperse the liquid collection or ameliorate the general condition of the patient, in my judgment the chance presented by the operation should be promptly given. There should be, of course, no misgiving as to the diagnosis where so formidable an operation is undertaken; yet it is not always easy to arrive at certainty on this head. Stokes says: "The diagnosis between mere dropsy of the pleura and the pericardium is not always free from difficulty."\* And, on the same subject, Trousseau declares that, "Notwithstanding the comparative certainty which has in our day been attained in the diagnosis of effusion into the pericardium, the diagnosis is still sufficiently difficult to leave room for the physician being deceived; and the special form of the dulness in pericarditis very different from the rounded dulness due to hypertrophy of the heart, is a very uncertain sign."† He then gives, by way of illustration of the difficulty mentioned, two examples of erroneous diagnosis of pericardial effusion by most competent physicians; namely, Dr. Vigla of the Hotel Dieu, and himself, in one of which the opera-

\* *Opus citat.*, p. 92, foot note.

† *Opus citat.*, vol. iii., p. 383.

tion was actually proceeded with to the stage of exposing the pericardium before the mistake was detected ; and in the other, although, according to his judgment, the operation was justifiable, he hesitated, and postponed its performance ; meantime the patient died. In the former of these cases great dilatation of the heart alone existed ; and in the latter, exaggerated hypertrophy with adhesion of the pericardium.

Extended area of percussion-dulness, dependent upon simple dilatation of the ventricles, would, however, in the great majority of cases, be readily distinguishable from that due to liquid in the pericardium, by the sharp, clear, and well defined character of the sounds of the heart ; whereas, in examples of liquid effusion, the sounds are masked, ill defined, and heard as from a distance.

Exaggerated dilatation is, moreover, usually associated with pulmonary emphysema and congestion of long standing, together with systemic venous engorgement and anasarca. It must, however, be admitted that the supervention of pericarditis with slight serous effusion in such a case, or the occurrence of simple passive dropsy of the pericardium, even to the amount of a very few ounces, so commonly associated with it in the advanced stage, may lead the most accomplished auscultator to diagnose copious liquid effusion exclusively.

For the differential diagnosis of cardiac hypertrophy, as usually presented, I would rely upon the prolonged and masked character of the first sound, as contrasted with the sharpness, clearness, and abnormal loudness of the second ; the heaving character of the impulse, and the displacement of the point of apex-pulsation. In hypertrophy, moreover, the sense of resistance to percussion in the seat of dulness is less than where it is caused by liquid effusion.

As in the example given by Trousseau, a thin stratum of liquid, the product of antecedent pericarditis or the result of passive effusion, occasional complications of cardiac hypertrophy from renal disease, interposed between the parietal pericardium and the heart, may so effectually mask the impulse and apex-pulsation as to render an accurate diagnosis difficult or even impossible. Such cases must be exceedingly rare, as I have met with only a single example.

In the progress of cardiac hypertrophy of renal origin, if the patient be not prematurely cut off by one or more of the other complications of renal disease, fatty degeneration of the heart will certainly ensue ; and, as a necessary consequence, its action will be so much enfeebled, and its sounds so masked, that the criteria just mentioned as between hypertrophy and hydro-pericardium, utterly and signally fail, and still more probably if slight effusion be superadded. In one such case (Case 29, Miss T.), I made the diagnosis of copious serous effusion into the pericardium, and on examination of the body after death, I found not more than two ounces of liquid in that cavity.

The site of puncture should be "of election," and determined mainly by two circumstances ; namely, the situation of maximum dulness and most distant cardiac sounds, on the one hand, and the absence, at the point contemplated, of the faintest approach to impulse, on the other. The fourth or fifth intercostal space, from one inch to two inches to the left of the sternum, has been usually selected for puncture, and consistently with the observance of the conditions above mentioned, will be found the safest and most convenient point to select. It should always be borne in mind, in selecting a point for puncture, that the heart may be attached to the pericardium at any part of its entire superficial area, and hence no absolute rule can be laid down, irrespectively of the two conditions above mentioned, for the guidance of the operator in the selection of the site of puncture. The avoidance of the internal mammary vessels renders it necessary to puncture not less than one inch from the edge of the sternum.

I would not contemplate the trephining of the sternum, as proposed by Laennec, nor the division of the eighth costal cartilage of the left side at its junction with the sternum, as recommended by Larrey. The former plan of proceeding would involve grave dangers other than those properly appertaining to the operation, such as emphysema of the anterior mediastinum, and through it, of the entire body ; osteo-phlebitis ; and caries of the sternum. The latter mode of operation would have the effect of weakening the entire framework of the chest, and would

by no means insure the object contemplated ; namely, the avoidance of the pleura.

For the performance of the operation, I should certainly give a preference over all other instruments to the pneumatic aspirator ; by the use of which the entrance of air into the pericardium and pleura would be effectually prevented, the escape of liquid into the cavity of the pleura avoided, and if, by misadventure, a wound were inflicted on the heart during the operation, it would be of so trivial a character, as to lead, in all probability, to no serious consequences.

Doctor Sibson recommends that a fine exploring trocar and cannula should be introduced obliquely upwards, either to the left of the xiphoid cartilage, or at the anterior part of the fifth intercostal space, and the fluid removed by means of a syringe applied to the extremity of the cannula.\* Thus he recognizes the advantages conferred by the application of the principle of suction in the performance of this operation, and I doubt not that he would now adopt, in preference, the modern and more perfect instrument for the application of this principle ; namely, the pneumatic aspirator.

In persons exhibiting the scorbutic diathesis in an aggravated form, as exemplified in sailors before the improvement of the navigation law, by which a penalty is attached to the non-shipment of fresh food and lime juice preparatory to a long voyage, the great serous cavities are liable to sudden and profuse extravasations of blood, which, acting as a foreign body, becomes a cause of acute inflammation of the most unfavourable augury.

*Scorbutic pericarditis*, so excited, is characterized by the suddenness of its invasion, and by the accompanying dyspnoea. The heart-sounds are faint and remote, and the area of precordial dulness is rapidly and greatly extended ; patients so affected rarely survive beyond the third day.

Karawagen, whose experience of the disease during an epidemic of scurvy among the sailors at Kronstadt, has been considerable, states that of sixty men who died of scurvy, thirty exhibited pericarditis of this form ; twenty-two pleuritis ; two

\* *Medico-Chirurgical Review*, July, 1854.



both pericarditis and pleuritis; and one peritonitis. The pericardium was thrice its natural size, and contained a bloody exudation amounting to four or five pints. No breach of vascular wall could be detected. The heart was reduced to one-half of its natural size, and both it and the parietal pericardium were covered with a red mould-like substance, which was readily removed by scraping with the edge of a knife. The substance of the heart was soft and fatty, and the endocardium was tinted red by imbibition; the ventricles were compressed and empty. Paracentesis was performed in two of these cases, in one of which it completely succeeded, the patient having been restored to perfect health after an interval of five months, although three and a-half pints of liquid had been removed from the pericardium.\*

Of this form of the disease, the differential diagnosis of which would manifestly depend upon the general evidence of scorbutus, the treatment should be mainly antiscorbutic, and should the physical evidence of sudden and copious effusion be presented, and accompanied by urgent dyspnœa and threatened failure of the heart, paracentesis would be indicated, as affording the only chance of saving the patient's life, and should be promptly performed.

Wounds of the pericardium are attended with considerable pain; although the heart itself is insensible to mechanical injury, according to Harvey, Ollenroth, and Bamberger. Georges Fischer gives fifty-one examples of wound of the pericardium, out of which there were twenty-two recoveries.† *Traumatic pericarditis* is always formidable, because of the all but necessary entrance of air into the sac, and consequent suppuration. The most active treatment should be promptly instituted, with a view to subduing the inflammation. Thus, leeching, mercury, and opium, should be at once directed, in proportion to the activity of the fever and the urgency of the symptoms.

*Tuberculosis* and *cancer* of the pericardium have been men-

\* "On Scorbutic Pericarditis and Pleuritis," *British and Foreign Medico-Chirurgical Review*, vol. xii., July, 1841.

† *Des Plaies du Cœur et du Péricarde*, *Archives Générales de Médecine* (Review), vol. i., p. 612, May, 1869.

tioned by Bonetus,\* Laennec,† Hasse,‡ Stokes,§ and Niemeyer.|| Tubercle, when present, is usually found in the pseudo-membranous product of chronic pericarditis, and associated with tuberculosis of the lungs, and with strumous enlargement of the mediastinal and bronchial glands. The tubercle is most frequently presented in the miliary form; but it may undergo caseous transformation, rarely, however, proceeding to suppuration.

Carcinoma of the pericardium is usually a secondary formation, and consecutive to cancer of the anterior mediastinum, or of the pleuræ; it is, in most cases, of the soft or medullary kind, and associated with turbid and flaky effusion into the pericardial sac.

But when the pericardium is the seat of cancer, that disease rarely succeeds ordinary pericarditis as a consecutive pathological transformation of exuded lymph; in this respect it differs pathologically from tubercle, which rarely appears within the pericardium, save as a new formation in false membrane.

The following cases will serve to illustrate the clinical history, pathology, and treatment of pericarditis.

CASE I.—*Chronic Endocarditis with Mitral Inadequacy; Acute Pericarditis with Effusion; Double Frottement; Death.*

Anne L., aged nine years, admitted into hospital, March 26th, 1866. Has had articular rheumatism, with swelling. A fortnight since was attacked with pain in the left side, accompanied with shortness of breath. At the date of admittance, the child was pale and fidgety; pulse 132, but regular; respiration 72. A double or "to-and-fro" friction-sound was audible over the lower part of the sternum, and a single systolic murmur at the apex. The latter was *slightly musical*, and audible likewise at the angle of the left scapula. Ordered, an opiate and mercurial plaster (Opium, gr. v, Emplast. hydrarg., 3iv, spread upon strong paper)

\* *Sepulchretum*.

† *Auscultation Médiate*, vol. ii., , p. 658.

‡ *Pathological Anatomy*, Sydenham Society's edition, 1846, p. 116.

§ *Opus citat.*

|| *Text-Book of Practical Medicine*, 1869, vol. i., p. 395.

over heart. *R.* Pulv. Ipecac. co., Pulv., Hydrarg. c. creta, aa. gr. ij. M. Ter die sumend.

March 29th. Pulse 126, steady, and of good volume; she is less choreic; "to-and-fro" sound audible at lower end of sternum, but soft; continue.

April 2nd. Dulness on left side to level of second rib, with absence of *frottement*; "to-and-fro" sound heard faintly, but more distinctly during pressure with the stethoscope. Patient fidgety, very weak and faint; pulse 126, and feeble. Wine to  $\text{ʒij}$ .

4th. Pulse 120, of good volume; precordial dulness of much less extent; "to-and-fro" sound now audible at apex; remarkably loud at base, and diffused generally over front of chest, but with greater distinctness upwards and to right side; continue.

5th. Pulse 132, and weak; respiration 54; increased extent of precordial dulness, with faint fremitus; "to-and-fro" sound not audible over apex, and less loud at base. Sinapism over heart, and continue. For several days past she has had herpetic eruption on lips.

6th. Pulse 120; respiration 42, irregular and suspirious; no friction audible over apex, but loud friction over base; no *frémissement*.

7th. Sat up yesterday; pulse 120, and much stronger; respiration 30; friction-sound diminishing, and audible only at base; precordial dulness less in extent; continue.

9th. Pulse 132; friction less distinct; systolic murmur audible at angle of left scapula; has bronchitis. *R.* Iodid. potass., gr. v, in decoction of cinchona thrice daily; a stimulating liniment to be applied over the precordium.

11th. Is very weak; pupils dilated; pulse 138; no cardiac impulse to be felt; there is dulness on left side from second rib downwards, and no friction is audible. Ordered, a blister to precordium, and tincture of digitalis,  $\mathfrak{M}\text{v}$ , in an ounce of decoction of scoparium every third hour; increase of stimulants.

20th. Precordial dulness is now of normal extent; "to-and-fro" sound is no longer heard, but over the apex of the heart a systolic bellows-murmur is heard with all its previous intensity; pulse 120, and full; tongue clean; appetite good. Patient has

been out of bed for the last three days, and during that time has been taking sulphate of quinia in gr.  $\frac{1}{2}$  doses thrice daily.

30th. Discharged to-day, restored to health in all respects, with the exception that, at the apex of the heart and in the left back, there is still audible a loud systolic bellows-murmur; precordial dulness normal; sleep and appetite unexceptional, and for several days past she has been taking exercise about the ward and corridor.

May 14th. She was readmitted to-day in a state of great debility, unable to stand without support; cardiac impulse was heaving; a double murmur was heard at the apex, and very indistinctly in the course of the aorta; pulse weak; cough, with expectoration; bilious vomiting. Blister to precordium, wine, chloric ether and spirit of camphor, lime water and milk.

17th. Is much relieved; stomach settled.

19th. On the evening of this day she became suddenly weak and agitated; breathing became embarrassed and gasping; she spat some blood, and continued to have fits of partial syncope every ten or fifteen minutes; the extremities were cold, and there was scarcely any pulse at the wrist; face pale and anxious; respiration loud, harsh, and rapid, and associated with bronchial râles on the left side, which was the only one admitting of examination, owing to the difficulty of moving the patient; she was unable to speak. Sinapism over heart, and a dessert-spoonful of brandy every second hour.

21st. She seemed moribund.

22nd. To my astonishment I found the patient, at my morning visit, cheerful; no longer suffering from dyspnoea or hæmoptysis; but the heart's action, as before, was associated with double murmur, and respiration was loud, harsh, and irregular.

23rd. Precordium prominent, and dull to an increased extent; capillary venous congestion over lower part of chest on both sides; cardiac impulse barely perceptible, and sounds scarcely audible. Blister over heart.

29th. From the date of last report patient was in the agony of death, up to 2 o'clock on yesterday (28th) when she died on being lifted out of bed contrary to my direction. The body and face



had become cedematous ; she had a teasing cough, and continued to expectorate small quantities of florid blood up to the period of her death. The heart-sounds had become inaudible, and the pulse imperceptible for several days. The body had become almost universally ecchymosed in broad irregular patches, separated by portions of unaltered skin. The trunk and thighs were most discoloured. The left side was clear, and yielded a loud and harsh breath-sound, accompanied with râles, whilst the right side, on which she principally lay, had become universally dull, with loud muco-crepitus of a ringing and gurgling character.

No *post mortem* examination of the body was permitted.

This case has been reported at length, but with all possible abbreviation, as a type of its class. It exemplifies the usual variation of symptoms having reference to the heart, and the corresponding physical changes discoverable in the precordium. There had been in the previous attack of rheumatism, endocarditis and valvular lesion, involving mitral inadequacy. The murmur due to the lesion was, in the second attack, which was accompanied with pericarditis, masked by the louder and more superficial sound of pericardial friction. On the temporary suspension of the latter by liquid effusion, the mitral murmur again declared itself ; it was followed by the usual consequences of mitral inadequacy, pulmonary congestion and hæmoptysis. The variations of friction-sound as to site, intensity, and rhythm, dependent upon corresponding fluctuations in the amount of liquid actually in the pericardium, are likewise well exemplified in the case.

#### CASE II.—*Acute Rheumatic Pericarditis ; Recovery.*

Kate K., aged sixteen ; admitted September 24th, 1866, complaining of pain in the hip and shoulder joints, and in the inter-articular portions of the limbs generally, which commenced a few days previously after exposure, whilst heated, to night air. Has had two similar attacks ; the first at the age of twelve years, after a severe wetting, and the other two years later, from exposure to cold. When admitted she complained of intense pain in the hips, shoulders, and knees, owing to which she found it

difficult to maintain the sitting posture even for a few seconds. Tongue moist and coated; copious acid perspiration; pulse full, and bounding; respiration 28, and interrupted; eyes brilliant, and right pupil dilated; great tenderness of the wrist joints, and general intolerance of pain; no cardiac symptoms, with the exception of sharp pain referred to the left mammary region. Ordered, alkaline mixture, composed of acetate and bicarb. of potash; and, to appease thirst, which was very urgent, the "imperial" drink, consisting of a dilute solution of acid tartrate of potash, lemon juice, and sugar.

26th. Pulse 108, and full; complains of acute pain in the left arm, and likewise across the front of the chest during inspiration.

27th. Pulse 103, full, but occasionally irregular; bowels confined. A plaster to be applied to the region of the heart, composed of mercurial plaster (3ss.) and powdered opium (gr. x.).

28th. Complains of pain over the heart; and, at the base, within a limited area about the diameter of a crown-piece, friction-sound is audible. Continue mixture.

29th. Precordial *frottement* audible over a larger area, but less loud than on yesterday.

30th. *Frottement* remarkably loud, and grating. Complains of pain at the heart, and want of sleep. Repeat plaster over heart, and let patient have gr. j. of watery extract of opium with gr. ss. of extract of colchicum every third hour, and alternately, and at equal intervals, half an ounce of lemon juice.

October 1st. Pains in the joints, for the last few days masked by the precordial distress, again constitute the chief cause of suffering. The affected parts to be rubbed with soap liniment, to which was added chloroform and tincture of aconite, each in the proportion of one-fourth.

2nd. Pulse 90, strong and regular; friction-sound very distinct.

3rd. Slept well last night; pulse 66; friction-sound of a much finer character; herpetic eruption appearing about the mouth and nose. Repeat pills.

4th. Pulse 78, regular; slight pain behind xiphoid cartilage;

slept well last night. *R.* Sulphatis quiniæ, gr. xxiv; Acid. sulph. dil., ʒj; Æther. chloric., ʒij; Aquæ, ad ʒviij. St. ʒss. 3tia q.q. horâ.

8th. Pulse 120; *frottement* not audible, and cardiac sounds masked; increased extent of precordial dulness. Emplast. vesicat., region. cord. applicand. Haustus sodæ et potass. tartratis.

10th. Febrile excitement has subsided; pulse 78; double friction-sound distinctly audible at base, and along the tract of the aorta. *R.* Hydrarg. perchlorid., gr. j; Decoct. cinchonæ, ʒviij. St. coch. mag. ter in dies.

On the following day the quinine mixture was resumed, and in the course of the next, the patient was discharged convalescent.

CASE III.—*Acute Pericarditis supervening on Aortic and Mitral valve-disease; Recovery.*

James W., aged seventeen, carpenter's apprentice, tall and delicate looking, was admitted to the Mater Misericordiæ Hospital, October 13th, 1866. One week previously he caught cold by exposure, and for two days subsequently had flying pains in the limbs, but no articular swelling. Two days later he was seized with acute pain in the region of the heart, which lasted till the day preceding that of his admittance, but had been, in some degree, mitigated by the use of sinapisms and warm applications. On the same day he began to suffer from dry cough, which was promptly brought on by assuming the recumbent posture. Patient is remarkably pallid and weak; pulse 132, and feeble; respiration 42; cardiac impulse visible in the sixth intercostal space, in a vertical line with the nipple; it is tumultuous and attended with fremitus. At the apex an accompanying systolic bellows-murmur is audible, preceded by friction-sound; and here the second sound cannot be heard. Over the entire basic region, but loudest at midsternum, and transmitted over front of chest generally quite to the clavicles, but not into the arteries of the neck, a double friction-sound is heard; it is likewise heard in the left scapular region; and in the infra-spinal fossa near the base of the scapula, a

double murmur of a cooing character is twice heard during each expiratory act, but ceases to be heard during inspiration ; and, on the anterior surface, a grating murmur, synchronous with the first sound, and quite superficial, is heard, with exceptional loudness during expiration, in the second intercostal space of the left side. Percussion and respiratory sounds normal over the entire chest ; tongue clean, and surface cool. Mercurial and opiate plaster to be applied over the heart. Perchloride of mercury, with compound tincture of cardamoms, to be given in infusion of calumba every third hour. To have gr. j of watery extract of opium at night, four ounces of wine, and good diet.

16th. Since last report the pulse has varied from 132 to 120 ; the latter being the rate to-day ; it is now dicrotous ; pupils small ; somnolence, thirst, and slight nausea, which latter may be due to the use of the expectorant mixture of the hospital for the purpose of alleviating his cough. Over the cardiac region generally, but most distinct at the apex, may be felt a well pronounced fremitus ; at the apex, and likewise at the base, a double *frottement* is audible ; but in the latter situation it is finer and *disintegrated* in character, by which it is at once distinguishable from double aortic murmur, likewise audible in the same situation, and from transmitted apex *frottement*. In both situations friction-sound may be readily intensified by gentle pressure with the stethoscope ; it is audible beneath both clavicles by transmission.

24th. Patient's condition much worse ; total loss of rest and appetite ; respiration greatly embarrassed, and 42 in the minute ; pulse varying from 120 to 132, weak, dicrotous, and slightly visible. At extreme left apex is heard a loud creaking systolic sound ; here second sound is soft ; pulsation of apex tremulous over a space of about one and a-half square inch, a short distance below and to the left of the nipple, and here *frémissement* is perceptible. At base, especially towards the right, a double murmur is audible, which, from its compound character, viz., being, on the one hand, *superficial*, *grating*, and *interrupted* ; and, on the other, *soft*, *continuous*, and *synchronous* with the cardiac sounds, I judged to be due in part to pericarditis, and in part to disorganization of the aortic valves. It was audible likewise



beneath both clavicles, but more distinctly so on the right side. Carotids pulsating visibly. Three days since mercurial inunction was had recourse to for a period of two days, in the proportion of 3ij daily. Draughts containing hydrocyanic acid, and chloric ether (℥ij and 3ss respectively) were given to allay irritability of stomach, and gr. j of opium at night to insure sleep.

26th. Is less restless; takes food well; at apex a soft systolic murmur is alone audible, but at base the compound double murmur may be heard; in left axilla a systolic murmur is faintly audible.

29th. There is physical evidence of congestion of the bases of both lungs, but this is more decided on the right side; patient passed a restless night; precordial dulness seems extended. To be dry cupped and poulticed over bases of both lungs.

November 1st. Pulse 126, and dicrotous; respiration 36; perspiration copious over head, neck, and chest; considerable increase in extent of precordial dulness, and less distinct impulse of heart; at apex a systolic murmur alone is audible, but it is likewise heard in left axilla; and at base a double murmur, but less loud than formerly. On yesterday (October 31st) patient complained of pain in right inferior lateral region, and here friction-sound was heard. Dry cupping and poultice to be applied at seat of pain.

6th. Has been sitting up for the last two days; slight laxity of bowels; mercurial to be stopped, and quinine to be substituted in gr. ij doses, ter in die, with ℥x of liquor of the pernitrate of iron.

7th. Feels and looks much better, and is taking more food; pulse 126, and of good volume; at apex of heart slight fremitus is perceived; and here a distinct double *frottement* is heard, masking the systolic murmur; double friction-sound is likewise audible at the base; but in the course of the aorta a double *bruit de soufflet* only; precordial dulness has become less in extent. The patient shortly afterwards left hospital convalescent.

The only remark I wish to make upon this case has reference to the modifying effect of respiration upon the friction-sound, as heard, at the date of the patient's admittance, in the second intercostal space, and at a corresponding point of the left back;

the friction-sound, which was probably due to both posterior and anterior pericarditis, was rendered inaudible by the expansion, and consequent intervention of the lung during inspiration.

CASE IV.—*Acute Rheumatic Pericarditis; Presystolic Friction-sound at Apex; Recovery.*

James D, aged 23, a butcher; admitted May 11th, 1869. On the 27th April he was attacked with pain in the knees, ankles, and wrists, but without swelling, save in the last named joints. On May 9th he experienced pain at lower end of sternum. When admitted he was pale and anxious looking; perspired somewhat; pulse 96; urine loaded with lithates; pressure over ensiform cartilage caused pain; and here, as likewise at the base of the heart, a "to-and-fro" friction-sound was audible; at the apex a single friction-sound was heard, which was presystolic in rhythm. R. Calomel. gr. ss; Pulv. opii, gr. ss. M. To be given in form of pill every second hour; poultice to precordium.

15th. Double friction audible throughout precordium, and transmitted to right second costal cartilage, but not into carotids; pulse 72; respiration 36.

20th. Pulse 120; friction-sound loud, double, and heard universally over precordium. St. Hydrarg. perchlorid., gr.  $\frac{1}{16}$ , 4ta. q.q. horâ.

22nd. Friction scarcely audible; precordial dulness much extended; pulse 108. Ordered: Emplast. vesicat. precord. applicand.; Mixt. alkal. diuretic.

23rd. Pulse 96; friction scarcely audible. To have porter and wine.

June 2nd. Since last report this patient has had recurrent attacks of pain and swelling of a migratory character in the joints. The liquid effusion has been slowly removed from the pericardium, and the friction-sound has again become distinctly audible at the base. At the apex, no friction or other abnormal sound has been heard for the last week. Patient has been out of bed for several days; he is now taking quinine, and recovering flesh. He was shortly afterwards discharged.

I will only remark upon the close resemblance, as to quality and rhythm, which the single friction-sound at the apex bore to the murmur of mitral constriction. This resemblance was further enhanced by the point (apex) at which alone it was audible. The distinction rested upon an occasional arhythmic irregularity of the sound, the readiness with which it was intensified by pressure, its *superficial* character, and the coexistence of double *frottement* over the right ventricle and at the base.

CASE V.—*Acute Rheumatic Pericarditis supervening upon Mitral and Aortic valve-disease ; Recovery.*

James D., aged thirty-four, porter, of temperate habits; admitted into hospital September 29th, 1869. Had rheumatism sixteen years ago, when the joints were swollen. Four months ago began to feel uneasiness about the chest; and five days previous to admittance was again attacked with pain in the joints.

State on admittance. Is anæmic; respiration normal; pulse 96, regular, and visible at the wrist; apex-beat one quarter of an inch inside nipple-line, and here a soft faint systolic murmur may be heard; second sound not intensified in pulmonary artery. At base a rough systolic, and a soft and faint postdiastolic murmur, were audible; both the latter were likewise heard in the course of the aorta, but the *former* only in the carotids. St. Citratis ferri, gr. v. 3tia. q.q. horâ.

About the middle of October, the man being then free from pain and fever, he was transferred to the care of my colleague, Dr. Cruise, in order to have a plastic operation done for hypospadias. Whilst under Dr. Cruise's charge he had a relapse of rheumatism, which was more acute than the previous attack, and was accompanied by pericarditis; a double but fine *frottement* was audible at the base of the heart; but, in order to educe it strong pressure with the stethoscope was necessary, and pain was then felt. St. Calomel., gr. j sing. horâ. Emplast. vesicat. precordio applicand.

In addition to the evidence of slight liquid effusion, afforded by the force of pressure required to bring out friction-sound, there was extension of the area of dulness, and faintness of the

cardiac sounds. Friction subsequently became again audible; on November 6th he was reported free from pain and fever, and at that date no *frottement* was anywhere to be heard. After a tedious convalescence he was discharged.

CASE VI.—*Acute Rheumatic Pericarditis and Myocarditis, with Pseudo-presystolic murmur; Mitral Reflux; Recovery.*

Lawrence O'B., aged twenty-four, paper manufacturer, temperate, admitted May 16th, 1868. On the second of May he was attacked with pain and malaise, and on the 9th was obliged to relinquish work. Has now slight pain and swelling in feet, hands, knees, and shoulders; pulse 144, weak, and somewhat irregular; apex-beat in usual situation, but extending a little to left; slight dulness over bases of both lungs, and here respiration is feeble; no cough. To have "imperial" for drink, and a pill every third hour, consisting of gr. ss of extract of aconite, and gr. iss of watery extract of opium; blister to right of midsternum where he complained of pain.

17th. Pulse 132, irregular, and full; perspires freely; sternal pain relieved by blister. At the apex of the heart a murmur is audible, presystolic in rhythm and rough in character; it was confined to the apex, left axilla, and back, in which latter situation it was very faintly audible.

19th. A distinct "to-and-fro" friction-sound audible at apex, where it is loudest, very superficial, and interrupted; it is likewise heard at the base, but less distinctly; no fremitus or increase of precordial dulness; pulse weak, failing, and irregular at wrist, where it cannot be counted; but the pulsations of the heart are 168 in the minute; respiration slightly hurried, but not embarrassed; no head symptoms; is pale; perspired copiously about head and chest last night, but feels much improved to-day. To have blistered surface dressed with mercurial ointment, and to continue pills.

June 10th. Owing to temporary illness which compelled me to relinquish hospital duty, I lost sight of the patient, having placed him under the care of my colleague Dr. Hughes. To-day I found that in the interim he had had double pleuritis with effu-



sion; right side still dull to a level slightly above nipple, and left side to a less extent; pulse rapid and feeble, but regular; heart acting at rate of 160 in the minute, and at the apex an ill defined "rub" precedes the first sound; elsewhere both sounds are sharp and clear; precordial dullness normal in extent; he is remarkably pale; and has been for some time taking iron under the directions of Dr. Hughes.

16th. Pulse steady, and 132; "rub" preceding first sound of heart has assumed still more the character of the murmur of mitral constriction. The patient has been out of bed during the last two days.

23rd. Pleuritic friction-sound on both sides; pulse 132, regular; is recovering colour; presystolic friction-sound at apex is very faint. Ordered: Infusi senegæ c. carb. ammoniæ.

30th. A faint pleuritic friction-sound is heard in the left lateral region; and preceding the first sound of the heart, at the apex only, a rough jarring murmur, which extends quite up to the first sound; impulse strong and extended, and presystolic *frottement* audible to right as far as middle line, but with decreasing distinctness. Discharged, and went to the country for change of air.

July 20th. Visited the hospital to-day; feels much improved; pulse regular, 120, and of moderate volume; apex-beat in nipple-line, and here, as also at left base, a single friction-sound of presystolic rhythm is audible. To have iron and chloric ether (of each ℥lxxx), in ℥viij of infusion of quassia, to be taken in ℥ss doses thrice daily; also cod-liver oil (℥ss) with syrup of iodide of iron (℥j), thrice daily after food. May resume light work, and take tepid baths. Discharged.

March 8th, 1870. Readmitted to-day; is now generally anasarcaous; swelling commenced in lower limbs and in face a month previously, accompanied by epistaxis and dyspnœa; a fortnight later swelling of genitals set in, as likewise cough and expectoration. Is now pale; face, lower extremities, and genitals much swollen; cough, and universally diffused râles; pulse regular, 96; apex-beat in nipple-line, and beneath sixth rib; impulse is diffused; a loud systolic bellows-murmur is audible at the apex; no intensification of second sound; urine contains

a good deal of blood, likewise albumen, and tube casts denuded of epithelium but presenting a few granular epithelial particles.

After active purgation with podophylline, hot and dry air-baths were given, with the effect of rapidly dissipating the dropsical effusion; the kidneys at the same time began to act vigorously, excreting about two quarts of urine in the course of the night, and of a more natural colour. In the course of a few weeks he was discharged much improved in general health, and quite free from dropsy.

The last attack was manifestly one of acute renal anasarca, the heart continuing to exhibit the traces of former endo-pericarditis. The pseudo-murmur at the apex might very readily have been mistaken for presystolic mitral murmur; but in this, as in all similar cases, the differential diagnosis was not only possible, but not difficult, quite irrespectively of all previous knowledge of the case, attention being given to the superficial localization and the *unsteadiness* of rhythm of the sound.

CASE VII.—*Acute Rheumatic Pericarditis; Mitral Regurgitation of non-organic origin; Recovery.*

Mary N., aged thirty, domestic servant, admitted December 8th, 1870, suffering from acute rheumatism engaging several joints of the lower limbs. Two days previously she complained for the first time of pain in the shoulders, arms, and hands. These joints became swollen; but in the course of the two succeeding days the disease shifted to the joints of the lower limbs, which, at the date of admittance, were swollen and tender. Perspiration copious; pulse 120, and full; tongue loaded and moist; face remarkably pallid. A faint thrill was perceptible with expiration at midsternum; and here, but not elsewhere, a sharp and single friction-sound, postsystolic in rhythm, was audible; action of heart abrupt. Alkaline mixture, and mercurial and opiate plaster to precordium.

13th. Friction-sound, double systolic and fine.

14th. Double friction-sound, viz., systolic and postdiastolic respectively in rhythm, coarser in character, and audible at base exclusively; pulse soft, regular, and 108; no carotid pulsation,

dyspnœa, or epigastric tenderness; slight increase in extent of precordial dulness, which extends vertically to one and a-half inch above level of nipple, and horizontally to right edge of sternum.

17th. Friction-sound of last mentioned rhythm coarse and audible at ensiform cartilage; precordial dulness less extensive; at apex first sound is rather prolonged; no carotid pulsation or epigastric tenderness. Has been taking for the last week a sleeping draught at night, composed of twenty to twenty-five minims of Battley's sedative, and four ounces of wine daily.

Two days since pain and swelling returned to wrists, right ankle being similarly affected.

25th. Dulness still extended somewhat beyond the limits of the superficial precordial region. Since date of last report pulse has varied from 108 to 120, but was full and regular; no precordial pain; respiration slightly quickened. On yesterday patient had a slight attack of dysentery, which still continues. A soft but indubitable systolic murmur is now audible at left apex. Alkaline mixture stopped, and an astringent ordered, consisting of aromatic chalk powder, tincture of catechu, and compound tincture of cardamoms.

27th. Dysentery still troublesome, chiefly in the morning; pulse 120; tongue dry and clean; breathing and pericardial dulness as at date of last report; at base of heart, and less distinctly at lower end of sternum, a sharp friction-sound is heard immediately succeeding the first sound; no other friction-sound audible, but at apex a distinct bellows-murmur exists, and extends into axilla. Emplast. vesicat. precordio applicand.

January 20th, 1871. First sound at base double; second sound sharp and clicking; no murmur audible anywhere; no systolic dimpling of surface, and no *frémissement*.

The *single* character of the friction-sound at the onset, and likewise at the close of pericarditis, the rhythm having been double in the interval, is noteworthy in this case. The development of a systolic murmur at the apex in the course of rheumatic pericarditis, might have been regarded as conclusive evidence of endocarditic complication; and, indeed, for some time I regarded it in that light. The subsequent history of the

case, however, supplies, in my opinion, a complete refutation of that view. A month subsequently, after a course of tonic and chalybeate medicines, the condition of the patient having in the interim undergone a decided general improvement, the murmur at the apex no longer existed. Where valvular inadequacy has once existed as a consequence of endocarditis, I cannot admit so complete a restoration of the valve to its pristine condition, as would preclude the continuance of reflux in some, however small, a degree. On the other hand, it seems not unwarrantable to conclude that, in a condition of anæmia and general debility, such as that witnessed in the course of slow convalescence of this patient, a state of atonicity or weakening of the muscular walls of the left ventricle of the heart would have occurred, in which, at the acme of systole, a yielding of some portion of the ventricular wall, under the centrifugal or counter-pressure of the contained blood, might have taken place, by which a valve-segment might have been retracted from the orifice through the medium of one of the papillary muscles and the corresponding tendinous chord, and regurgitation as a consequence have taken place. Improvement in the general condition of the patient under tonic and restorative treatment would, in that view, afford an adequate explanation of the cessation of murmur. It is noteworthy, that in the progress of recovery and cessation of this murmur, reduplication of the first sound at the apex was observed, constituting, no doubt, a stage of redux transition from systolic murmur to a normal first sound.

CASE VIII.—*Hypertrophy and Dilatation of Right Ventricle ; Partial Hypertrophy of Left Ventricle ; Chronic Non-rheumatic Pericarditis ; Death.*

John S., aged about forty-five, van driver, admitted January 17th, 1871. Has been short-breathed since childhood, and spat some blood a few months ago. Two months prior to admittance feet and legs began to swell.

When admitted he was suffering from aggravated dyspnœa, being unable to lie down ; the face livid, and the cervical veins distended to the utmost degree, but not pulsatile. The lower



limbs generally were cedematous, tense, and livid; the genitals also swollen; radial pulse slow, soft, large, and regular. There was physical evidence of aggravated pulmonary emphysema and bronchitis, with congestion and œdema of the bases of both lungs.

Cardiac impulse was felt at the ensiform cartilage, where both sounds of the heart were distinctly audible, the first being rather sharp and clear. There was likewise audible in this situation an equivocal murmur of a harsh and somewhat grating quality, accompanying the first, and also, but very faintly, the second sound. The cardiac sounds were elsewhere less distinctly audible, and free from murmur. The urine was of normal specific gravity, and contained a trace of albumen. *R.* Tincturæ digitalis, Spirit. æther. nitr., aa. ℥x, in camphor water, every third hour; counter-irritation to chest, and a moderate allowance of whiskey punch. On the following day the pulse was irregular, but otherwise there was slight improvement. On the 19th he was incoherent; became delirious; and died comatose on the morning of the 23rd.

*Post mortem* examination of body. About thirty ounces of serum were found in each pleural cavity; the lungs were generally emphysematous, and congested at the base, where a few nodules of sanguineous extravasation were found. About three ounces of serum, but no fibrinous flakes, were found in the pericardium. There was a large "milk spot," two inches in diameter, on the anterior surface of the right ventricle, and on its apex a rough patch of exuded lymph; the free surface of the pericardium, to the extent of about one and a-half inch in diameter, was rough and jagged; the serous investment of the right auricle and appendix generally was likewise rough and opaque, and that of the left auricle also, but to a less extent. The heart weighed twenty and a-half ounces; it was globular in figure, and was formed in greatest part of the right ventricle, of which the apex was entirely composed. The right chambers contained some partially decolorized fibrin, which quite filled the auricular appendix and overlay the orifice of the pulmonary artery. Both these chambers, in the remaining portion, were full of dark coagulated blood. The right auricle was dilated, and the right ventricle greatly dilated and hypertrophied; its wall, midway

between the base and the apex, was three-eighths of an inch thick. The tricuspid opening freely admitted the four fingers and thumb to the roots. Both the tricuspid and right semilunar valves were normal; the left auricle was unaltered; the left ventricle somewhat hypertrophied; mitral and aortic valves normal and competent.

Several days anterior to death, the diagnosis was made of pericarditis, cardiac hypertrophy and dilatation, pulmonary emphysema, bronchitis and cedema, and absence of valvular disease.

In a state of extreme engorgement of the right chambers there is no murmur, even though there be tricuspid reflux, owing to permanent distension and enfeeblement (*asystolia*) of the right ventricle. It is in cases of tricuspid valve-disease, where weakening and distension of the right chambers do not exist, that murmur at the tricuspid orifice is developed.

CASE IX.—*Mitral Regurgitation, and Hypertrophy, of some years' standing; Recent Pericarditis and Pneumonia; Death.*

William H., aged eighteen, shop-assistant, admitted March 17th, 1871. Eight days previously, after a walk, he was seized with severe pain across the front of the chest and cough accompanied with blood-stained sputum. Has never had rheumatism, but was regarded as a delicate person. When admitted he was in a state of collapse, the pulse being entirely suppressed in the right radial, and very feeble and quick in the left radial artery; surface cold; cheeks, lips, tip of nose, external ears, and finger-ends were quite livid; respiration quick, shallow, and painful. Precordial dulness was not perceptibly increased in extent; cardiac impulse strong and double; the second sound was double, the first element accompanying the second or diastolic impulse, and everywhere loudest; apex-beat in nipple-line and fifth intercostal space, and here a blowing, substitutive, systolic murmur was audible, and strictly limited to area of apex. At the date of admittance this murmur was not audible, but was detected on the following day; on the second day it was again inaudible; but on the third day (20th), and subse-

quently, up to the close of life, it was distinctly heard. Hepatic dulness extended four inches below costal cartilages, and there was tenderness at the epigastrium. The lower and posterior portion of the right side was dull, and bronchial respiration and crepitus were audible over the posterior right base. Sputa viscid and amber-coloured. *R.* Liquor. Hoffman.; Spirit. æther. nitr., aa. 3ss; Tincturæ digital., ℥v, cum aqua camphoræ, ut ft. haustus. 3tia, qq. horâ sumend. Whiskey and water frequently in small quantities, and dry cup and poultice right side at seat of dulness.

18th. Pulse stronger; face now of a crimson hue; moans with pain referred to a point one inch below and to inner side of nipple; double impulse very distinct; one (the stronger) accompanying the first sound of the heart, the carotid pulse, and the apex-murmur; the other, less strong, but decided, and everywhere synchronous with the first element of the double second sound. Cupping to four ounces over right base of right lung, and two leeches at site of pain in precordium, to be followed by a warm poultice. *R.* Calomel., gr. ss; Pulv. Jacobi, gr. j. *M.* Ft. pulv. quinta. Una qq. horâ sumend.

21st. Has now taken twenty-two powders, and exhibits the early signs of mercurial action; is quite relieved from pain; respiration easy; pulse on left side 108, and strong, but imperceptible on right side. *R.* Sulph. quiniæ, gr. ij; Sulph. morphiæ, gr.  $\frac{1}{16}$ . *M.* Ft. haustus. 3tia qq. horâ sumend. Poultice to side to be continued.

22nd. Slept well, and is much improved. To have a chop.

April 3rd. A few days since, the base of the left lung was attacked with pneumonia, for which he was cupped and blistered. The sputa were for some days deeply tinged with florid blood; respiration much embarrassed, and the pulse on two several days registered 192. This morning there is sharp diarrhœa; respiration 40. The left pleural cavity was found nearly full of liquid, dulness extending within a short distance of the clavicle; the upper portion, however, to the extent of a hand's breadth vertically, both before and behind, yielded muffled tympanitic resonance. Respiratory sound was nearly suppressed, but not altered in character over the entire left side; ægophonic voice was

audible beneath the spine of the scapula; and vocal thrill was entirely abolished on the left, whilst it was distinctly perceptible on the right side. The heart was displaced to the right of the sternum, and pulsated in the right fourth intercostal space, half an inch inside the nipple-line. The first sound was free from murmur; the second faintly double, its first element being accompanied as before by diastolic impulse. Puerile respiration is audible over the entire right side, which is clear on percussion to middle line of sternum, beyond which absolute dulness exists, modified superiorly by muffled tympany as already stated.

R. Tinct. digitalis, 3j; Spirit. æther. nitrosi, 3iv; Sp. juniper., 3ij; Decocti scoparii co., ad 3xij. St. 3j 3tiis horis. Whiskey in small doses, and frequently.

April 8th. Died suddenly last night, with symptoms of syncope.

*Post mortem* on the 9th. About eight ounces of serum in sac of pericardium, which was not attached to heart, or lined by false membrane, but thick, opaque, and perfectly smooth throughout. Heart somewhat enlarged, weight eleven and a-half ounces; left ventricle hypertrophied and dilated; it was half an inch thick except at apex, which was very thin; structure of heart and valves absolutely healthy. A yellow fibrinous thrombus extended from the right auricular appendix into the right ventricle, but without blocking the tricuspid orifice, or entangling the valve. The exterior of the pericardium on the left side was thickly covered with miliary tubercle of the size of duck-shot; it was blood-stained, and firmly attached to the inner surface of the left lung, which presented a similar appearance. The left lung was covered with false membrane, reduced in volume, and carnified, with the exception of the apex, which was attached to the chest-wall, and contained some air; in the base of this lobe there was a nodule of sanguinous extravasation, and in its centre a caseous mass as large as a hazel-nut; a few caseous masses of smaller size existed in the apices of both lungs. The lower lobe was nowhere adherent, and lay across the spinal column, its inner surface being attached, as already stated, to the pericardium, which lay to the right of the middle line; the base of the heart corresponding to the right fourth intercostal space in



the nipple-line, and the apex on the same level, but internally to the base. The areolar tissue of the anterior mediastinum was of a gelatinous character, and infiltrated with serum. The right lung was partially attached by its anterior surface to the chest-wall; the base was dark and congested, but resonant. Several large lymphatic glands of stony hardness surrounded and compressed the right subclavian artery; the bronchial glands were likewise enlarged. Several pints of dark blood-stained serum were found in the left pleura, displacing the pericardium to the right side.

In relation to double cardiac impulse, I have already expressed my opinion, that the second shock is due to the abrupt expansion or diastole of a dilated and hypertrophied left ventricle. In the case above narrated, the first and stronger impulse coincided with carotid pulsation, and preceded, by a very slight but appreciable interval, that of the radial artery; it was, therefore, the representative of the normal impulse of the heart; the second and weaker impulse was no less certainly ventricular, because felt in the same situation as the first, viz., at and immediately above the apex only; and diastolic, because coincident in time with the first and louder element of the double second sound, which I have already shown to represent the normal second sound. In reference to the doctrine of Hope, implying a necessary dependence of double shock upon adhesion of the pericardium, as the starting point of the pathological series issuing in hypertrophy of the heart, and as illustrating the error involved in that doctrine, the case is likewise interesting. No adhesion whatever existed, and yet double impulse was well pronounced, and associated with dilated hypertrophy of the left ventricle exclusively.

Temporary and fluctuating mitral reflux murmur I can attribute only to partial yielding of the wall of the left ventricle in systole, and consequent displacement of the valve. It might be supposed due to localized friction on the left side and exterior of the pericardium, roughened by tubercular deposition and recent exudation of lymph; but in that case its rhythm would have been in some degree respiratory, its quality in all probability harsh, its regularity much deranged, and its seat of greatest in-

tensity more remote from the mesian line. Yet, I am willing to confess the existence of some doubt in my mind that it was not caused by exo-pericardial friction, because of the actual firmness of the ventricular walls as determined by the autopsy, and the final cessation of the murmur on the third day after admittance.

Localized tubercular deposition on the exterior of the pericardium and corresponding portion of the pleura, is not common, and is worthy of being recorded as the possible cause of an acoustic phenomenon liable to be confounded with endocardial murmur. Suppression of either radial pulse, whilst the other is perceptible, will be found due, in ninety-nine out of one hundred cases, to the pressure of an aneurism, or of enlarged lymphatic glands in the neck or axilla. If, therefore, aneurism be excluded, the diagnosis of glandular pressure upon the main artery of the limb may, with equal confidence, be made.

CASE X.—*Circumscribed Pericarditis confined to the Root of the Pulmonary Artery.*

— Y., a girl of sandy complexion, aged about twenty, admitted in May, 1871. Has had rheumatism, and at the date of admittance suffered from that disease in a sub-acute form, the attack having commenced three weeks previously. The left hand and forearm were stiff and wasted, and there was considerable tumefaction, without tenderness, over the left internal condyle of the humerus. There was slight pain and swelling of the joints of the right arm; scarcely any febrile action; pulsation and sounds of heart normal, with the exception that, over the inner portion of the left third intercostal space close to the sternum, a harsh grating sound was heard accompanying the first sound of the heart; it was strictly limited to the situation mentioned, audible over a space not larger than the bell-end of the stethoscope, intensified by pressure, and barely audible when pressure was not made. There was no murmur at the right base, or in the neck.

After a residence in hospital of about a month's duration, she left on the 1st of June, improved in general health, and free

from pain, but without any alteration having taken place in the character or intensity of the murmur.

In addition to "auricular pericardial friction,"\* characterized by *frottement* of presystolic rhythm, and limited to one or both auricles, basic pericarditis, without implicating the ventricles proper, may likewise yield an attrition-sound simulating endocardial murmur. In such case the friction-sound is strictly systolic in rhythm, and due to pericarditis involving the roots of the great arteries exclusively. It is remarkably persistent.

The case just narrated was an example of this kind; the effused fibrin having been limited to the root of the pulmonary artery.

#### CASE XI.—*Pericarditis with Effusion ; Recovery.*

Margaret B., aged fifty, admitted October 8th, 1867; health good up to the preceding summer, when, after the sudden retrocession of a cutaneous eruption (kind not known) she began to suffer from palpitation. A month previous to admittance she was attacked with pain in the left side, accompanied with difficulty of breathing; swelling of feet and abdomen subsequently took place.

At the date of admittance the feet and legs were much swollen and slightly congested; there was some liquid in the peritoneum, and a good deal of subcutaneous serum; pulse scarcely to be felt at wrist, but counted by heart it registered 138; cardiac action feeble and irregular, and no impulse perceptible; sounds masked; precordial dulness extended to the left one and a-half inch outside nipple-line, to the right edge of the sternum, and upwards one and a-half inch above level of nipple; respiration embarrassed; cough, with mucous expectoration. St. Calomel., gr. ss, secunda q. horâ; blister to precordium, and chloric ether (3ss) in camphor water every third hour; wine. Dorsum of left foot punctured to allow escape of serum.

14th. Pulse (counted by heart) 72, very weak, and imperceptible at wrist; precordial dulness less extensive towards the left, and extending on right side only to middle line; cardiac impulse now perceptible; both sounds distinctly heard, and a rough,

\* Hyde Salter, *Lancet*, July 29th, 1871.

jarring, and superficial quasi-murmur with the first sound is occasionally heard over the base, loudest to the left of the middle line, and faintly audible at the apex.

15th. R. Emplast. hydrarg., ʒss; Pulv. opii, gr. x. Ft. emplastr., super cord. applicand.; stop pills, and continue mixture; puncture on dorsum of left foot discharging serum freely. Returned home, at her own desire, the pulse then being intermittent and irregular, and the cardiac phenomena as previously described.

CASE XII.—*Chronic Pericarditis and Pleuritis; Suppression of Right Radial Pulse; Death.*

James F., aged thirty-eight, bricklayer, admitted February 10th, 1871. Has had occasional hæmoptysis during the last six years; breathing has been short the last two years; has drunk hard. On the 1st February began to experience a sensation of fulness and dragging in the lower part of left side; this continued up to the 7th inst., when he was suddenly attacked with dyspnoea of an urgent kind. The paroxysm soon subsided, but his breathing continued much oppressed to date of admittance. Breathing was then greatly embarrassed, thirty-six in the minute; thoracic movement free and equal on both sides, but supra-sternal and left supra-clavicular space sank in with respiration. Resonance was normal on both sides; and on right side respiratory sound was loud and harsh, with sparse râles, but on left it was everywhere feeble, and in infraclavicular region almost *nil*. Pulse 120, full and regular on left, but on right side it was scarcely perceptible; the artery was tortuous, and the fingers clubbed.

March 5th. There is now general bronchitis, with *gargouillement* in left front and in right posterior base. Friction-sound has existed for some weeks past over lower left side, and to-day it is audible over left boundary of pericardium, where it coincides with both respiration and cardiac impulse, but only in the recumbent posture. For some weeks before death the left side was absolutely dull, and respiration was faintly audible only behind. Died, April 26th. About four pints of blood-stained



serum were found in the left pleura; the left lung was enveloped throughout in a thick layer of false membrane, and was adherent to the pericardium, but nowhere to the chest-wall. The lung was reduced in volume and partially carnified, and in its apex were some masses of crude tubercle, and a smooth-walled cavity the size of a filbert. With this cavity no bronchial communication could be traced, nor did water injected into the principal bronchus find entrance into it. The right lung was universally adherent; the pericardium greatly thickened and rough on its inner surface; it was incorporated with both pleuræ by false membrane half an inch thick. In the pericardial sac was a pint of amber-coloured serum. The surface of the heart and the roots of the great vessels were completely enveloped in false membrane of a reticular and granular character, and two to three lines thick. The heart, which was pointed at the apex, weighed fourteen and a-half ounces; the outer surface of the right ventricle, to the depth of one half its thickness, was composed of pure adeps; its muscular structure was pale and everywhere granular, and no striæ were visible. The left ventricle was half an inch thick, and dilated; the aorta was slightly atheromatous in a few spots; valves all healthy; some flakes of fibrin were found in the right ventricle, extending into the pulmonary artery. In the recumbent posture the left lung advanced as far as the investing false membrane allowed, and by approximating the pericardium to the heart, became accessory to the friction-sound which accompanied both the movements of the heart and of the lungs. The extra weight of the heart was in greatest part due to the thick layer of false membrane by which it was enveloped; and the great respiratory distress exhibited during the last weeks of life was in a great degree due to fatty transformation and failure of the right ventricle.

I regret that no examination of the right supraclavicular region was made, with a view to ascertaining the cause of suppression of the right radial pulse.

CASE XIII.—*Rheumatic Endo-pericarditis; Recovery.*

Jane A., aged twelve years, admitted October 31st, 1870, for subacute rheumatism; ill a week before admittance. When she

was first examined a suspicious prolongation of first sound was detected at the apex, which, after a few days, assumed all the characters of a murmur.

November 20th. Apex-murmur, distinct, systolic, and soft; double pericardial friction-sound over precordium generally, extending upwards to left clavicle, but not heard at apex, the second element being postdiastolic; pulse 108; no pain in joints, or over heart.

25th. Both elements of friction-sound synchronous with sounds of heart.

29th. One friction-sound only audible at base, systolic in rhythm, and harsh in quality, but *not* audible at right second costo-sternal joint, nor in the carotids; no fremitus. Is convalescent, and taking iron and quinine.

30th. No basic murmur whatever audible, but at left apex a distinct soft systolic murmur exists. Discharged.

CASE XIV.\*—*Rheumatic Pericarditis engaging especially the Root of the Pulmonary Artery; Tubercular Softening of Left Lung; Recovery.*

Peter D., aged twenty-two years, carriage-smith; had rheumatism two years anterior to date of admittance on the 15th of February, 1869. Taken ill eight days previously with pain in knees and general muscular tenderness. When admitted, his wrists were swollen and red; perspired freely; heart's action obscure; sounds muffled and of a suspicious character. St. Lemon-juice,  $\bar{3}$ ss, 3tia q.q. horâ.

20th. Pulse 132, full and dicrotic, but soft; respiration 72. Last night he was attacked with pain "in the stomach," which, doubtless, was precordial, and was relieved promptly by the application of two leeches by the resident pupil, Mr. Kelly. Precordial dulness now extended, and sounds scarcely audible at apex. At left base a faint friction-sound exists. To have calomel, gr. ss, with James' powder, gr. j, every hour; and

\* This, and all subsequent cases of pericarditis published in this work, came under the notice of the author after the list from which his averages were deduced was made out, and, consequently, are not included in it.

drachm-doses of spirit of nitrous ether and spirit of juniper, every second hour.

21st. Diarrhœa; pulse 132; respiration 48; extended precordial dulness, and no friction-sound audible; bronchitis, and congestion of posterior bases of both lungs. Mercury to be stopped, and patient to be dry cupped behind. To have thrice daily, in mixture, gr. iij of compound powder of chalk, with ℥ v of tincture of opium.

23rd. Pulse 132; respiration 60; heart-sounds obscure and weak. To have gr. iij of citrate of iron and quinine thrice daily; wine and porter.

26th. Pulse 108; respiration 48; over left third intercostal space near the margin of the sternum, *i.e.*, over root of pulmonary artery, there is a "to-and-fro" friction-sound, each element being double, and counting 156, whilst the radial pulse was only 108; one inch lower down, at level of nipple, only a single rub is heard, which is synchronous with the radial pulse.

Friction was suspended on the 27th, but was again heard on the 28th, and on the 29th it was masked by loud râles. On that day the patient was very weak and nervous; breathing greatly embarrassed; both bases dull posteriorly. A large blister was applied here.

March 3rd. Pulse 120; respiration 60; cough troublesome, and expectoration thin, frothy, and slightly tinged with blood; both sides comparatively dull behind, with a peculiar tympanitic note below angle of left scapula, over a space of a hand's breadth.

4th. Pulse 108, soft, full, and compressible; respiration 36; less cough; loud gurgling râles in upper left front.

8th. General softening of left lung; apex-beat in fifth intercostal space, half an inch outside nipple-line; pulse 96; slight hectic flush, and perspiration at night; expectoration frothy and mixed with blood.

11th. Pulse 96; no perspiration; less cough; respiration 24; râles and dulness above nipple diminished; apex-pulsation in nipple-line and fourth intercostal space; small purpuric spots on feet and legs; has been out of bed for the last two days.

April 8th. Slight cough, with night sweats ; breathing quick ; tongue clean, and appetite good ; has gained flesh.

In reference to the subject under discussion, the most noteworthy feature in this case is the multiplication of friction-sounds in the site of the pulmonary artery, a consequence, no doubt, of the undulatory movements of that vessel in alternate distension and reaction ; and this, whilst one inch lower down a single *frottement* only existed, which was synchronous with the first sound of the heart.

CASE XV.—*Renal Disease ; Chronic Pericarditis ; Cardiac Hypertrophy and Hepatic Congestion ; Death.*

John C., aged twenty-eight years, brewer's carter, of sandy complexion, admitted September 19th, 1870. Fourteen weeks previously his feet became swollen, and a fortnight later he had palpitation, and he noticed enlargement of the liver. Three days before admittance he had bilious vomiting and coughed up some florid blood, and on the day of admittance he had a rigor. Never had rheumatism. When admitted he was pale, and unable to retain food or drink of any kind. The vomited matter was tinged with bile. Pulse 84, regular, and very weak ; feet swollen ; urine 1·02, containing lithates, but no albumen ; hepatic dulness extended down nearly to the umbilicus, and upwards to the level of the fifth rib ; epigastric hollow obliterated, and hepatic surface smooth, but very tender to pressure ; no discoverable splenic enlargement ; urgent dry cough, by which pain in region of liver is aggravated. The apex of heart pulsated two inches outside the nipple-line, in the sixth intercostal space ; and here, for a period of two days, a *bruit de cuir neuf* was heard, which on the first day was systolic, and on the second day diastolic in rhythm. Small doses of calomel and James' powder were given, but promptly rejected ; leeches and poultices were applied at the epigastrium ; wine, beef-tea, and ice were administered. Much relief was experienced from the leeching.

For a period of about ten days the friction-sound now ceased to be audible. On October 2nd it was again heard, and was



then systolic in rhythm; and on the following day, whilst the apex *frottement* preserved the same rhythm, a double friction-sound of a decided character was audible over the remainder of the precordium.

The general condition of the patient was now improved; pulse regular, stomach less irritable, cough nearly gone, and a few râles only audible. A mercurial and opiate plaster was applied to the precordium.

5th. A fine systolic *frottement* exists at apex, but is not audible in axilla; elsewhere in precordium a rough double friction-sound is heard; œdema of feet, legs, and scrotum.

16th. Since date of last report the prepuce has been repeatedly punctured to facilitate micturition; the pulse varied much, occasionally intermitting; the stomach continued irritable, and the precordial sounds varied little. On this day, however, cardiac action and sounds being normal, no attrition or other sounds are anywhere audible; precordial dulness of normal extent; and stomach capable of retaining solid food.

18th. The phenomena of cardiac action are remarkable; both sounds are seemingly reduplicated, so that four sounds, occurring in rapid succession, correspond to a single beat of the radial pulse, which is regular, of moderate strength, and eighty-four in the minute. These reduplicated sounds are in some degree masked, but they mimic the normal sounds of the heart, and are audible throughout the precordium. (*Bruit de rappel.*)

On the following day (19th), only three sounds were audible, the first two of which corresponded in rhythm with the cardiac impulse, and represented the normal first sound; and on the 20th, the sounds were again only two in number, and of the character exhibited before the 18th, *i.e.*, slightly dulled but otherwise normal. The patient had a sensation of "dragging" at the apex with each action of the heart. Digitalis was now given in  $\mathcal{M}$ x doses of the tincture every third hour, with an equal quantity of spirit of nitrous ether. Urine increased in quantity, and œdema of genitals and of lower limbs decreased; cough had nearly ceased, but some bloody sputa had appeared.

26th. Great œdema of left arm and hand, and of lower limbs; pulse 108 and weak, but regular; congestion of posterior base of

right lung; sputa bloody, and patient somnolent, but hopeful. A loud *bruit de cuir neuf* is audible all over precordium, also beneath both clavicles, on both sides of back, and along spine to lower dorsal region; its rhythm is double, one portion being synchronous with the impulse and first sound, and the other posterior in time to the second sound, *i.e.*, one was systolic, and the other postdiastolic. *Frémissement* perceptible all over precordium, and a tremulous movement of the intercostal spaces to some extent visible, corresponding in time with the double *frottement*. There is no retraction of the epigastrium or intercostal spaces, and no distention of the jugulars, or venous collapse in the neck.

27th. The patient complains of acute pain in the region of the heart; at the base a loud harsh double *frottement* is audible, and at the apex a double attrition-sound of similar rhythm, but higher pitch, and musical quality. Precordial pain relieved by a leech, from the bite of which there was rather copious bleeding, and on the following day the patient was very weak; pulse 96, regular, and feeble; cardiac action likewise feeble, and friction-sounds less loud but otherwise unaltered, and still faintly audible under both clavicles. Œdema of genitals and lower limbs now extreme, and seems likely to give rise, by tension, to gangrenous inflammation and sloughing of the integument of the thighs.

28th. Punctures freely made, from which there was a copious flow of serum. The haggard and wasted aspect of the face now characteristic; breathing much oppressed, and stomach occasionally rejects food and drink. Death occurred Sunday, October 30th.

*Post mortem* examination twenty-four hours later. There was thickening and opacity of the peritoneum, but no ascites. The capsule of the liver was thickened and white over a space of one and a-half inch in diameter at each side of the suspensory ligament. The liver was somewhat enlarged, but otherwise unaltered. Each pleural cavity contained about a quart of clear serum. The lungs were sound, with the single exception that the lower lobe of the right was congested and œdematous. In the pericardium about six ounces of viscid amber-tinted liquid were found, which,

on microscopic examination, exhibited blood-corpuscles and compound granular cells. The heart was globular in figure, and hypertrophied; it weighed eighteen ounces. The left ventricle was much hypertrophied, and somewhat dilated, its walls below the base being more than half an inch thick. The right chambers were, as to capacity and thickness of walls, strictly normal; valves everywhere normal and competent. On the anterior surface of the right ventricle a patch of rough fibrinous deposit was found: it was three-fourths by half an inch in diameter, and coarsely striated at an acute angle with the long axis of the heart; and on the parietal layer of the pericardium a similar patch, corresponding in situation and extent, existed. On the posterior surface of the heart two patches of the same character were found; one upon the visceral, and the other on the parietal pericardium. A large patch was likewise found on the right auricle; and another, but smaller, on the left apex. In these several situations flakes of white and partially attached fibrin depended from the surface of the heart. The fibrin was everywhere white and skein-like, and entirely devoid of blood-vessels or spots; it loosely adhered to the heart, and, microscopically examined, was filamentous in structure. Kidneys small, granular, and pale; on section, cortex was thick, and presented excess of fibrous structure, with much fat in granules and large masses; surface of section was generally pale, and pyramids reduced in size.

This case well illustrates the unsteadiness of the abnormal sounds, as to number, rhythm, and site. The loudness and the extensive diffusion of the *frottement* in connexion with hypertrophy of the heart; and the blanched appearance, low organization, and non-adhesive character of the effused lymph, distinctive of chronic pericarditis of renal origin, are likewise noteworthy.

CASE XVI.—*Circumscribed Pericarditis engaging the Root of the Pulmonary Artery only; Persistent Frottement; Recovery.*

Mary A. K., aged thirty-two years, admitted into hospital, January 13th, 1872, for mild rheumatism engaging the smaller joints of feet and hands, and attended with perspiration and vascular excitement. Had a similar attack twelve years pre-

viously. Two days after admittance a faint double friction-sound was heard over the root of the pulmonary artery; it was not attended with *frémissement*, and was inaudible elsewhere. The patient was restored to perfect health in the course of a fortnight, under alkaline treatment; but the friction-sound in the original situation, to the left of the sternum, continued unaltered at the date of her discharge.

CASE XVII.—*Circumscribed Pericarditis; Recovery.*

Kate M., aged fourteen years, admitted February 7th, 1872. Two days before, she complained of pain in the feet, and on the date of admittance, in the right hand, the fingers of which were swollen. Pulse 96, regular; pericardial systolic *frottement* confined to root of pulmonary artery, and accompanied with feeble fremitus at acme of expiration. Discharged cured after a few weeks' residence, the pericardial attrition-sound having continued unaltered up to that date.

CASE XVIII.—*Rheumatic Pericarditis with Effusion; Recovery.*

James F., aged seventeen years, admitted under Dr. Nixon, November 17th, 1871. At Doctor Nixon's request I examined the patient, and, by his kind permission, learned the following particulars respecting him. He had always been delicate, and had rheumatism three years previously; was again attacked with pain and swelling of the small joints of the feet a few days prior to admittance. The disease, as commonly happens in such cases, migrated to the hands, and the patient was suffering acutely when I visited him with Dr. Nixon. The pulse was rapid, but regular; the superficial epigastric veins engorged and tortuous, but no venous congestion of the neck; precordial dulness normal in extent; the heart's action tumultuous, but its sounds normal, with this exception, namely, that in the second intercostal space of the left side, close to the sternum, and over a space equal in size to the bell-end of the stethoscope, a sharp grating noise accompanied the second sound; a fremitus was likewise perceptible in the same situation; but neither phenomenon was elsewhere discoverable.



Having never previously heard a single pericardial friction-sound of *diastolic* rhythm, I expressed the opinion that the sound in question was an example of the exceedingly rare phenomenon, a regurgitant murmur in the pulmonary artery.

In the course of a few days loud and characteristic pericardial friction-sound of double rhythm was audible throughout the precordium, and no doubt could exist as to the nature of the case. Effusion rapidly set in, and the patient for several days was on the point of death; but, under the judicious treatment of Dr. Nixon, with a mild preparation of iron, and an ample allowance of wine, the liquid effusion was gradually dispersed, the double *frottement* returned, and ultimately ceased, the superficial venous engorgement of the abdomen disappeared, and the patient, restored to comparatively good health, was discharged.

I did not at the time venture upon an explanation of the localized venous engorgement of the abdominal wall; nor have I since been able to suggest a plausible solution of the difficulty. The strictly diastolic rhythm of the localized pericardial friction-sound at the outset, constitutes, however, the principal feature of interest in this case; it is the only example of the kind I have met with. The error of diagnosis arising from the rarity and eccentric character of the phenomenon, which the harsh quality of the sound should have prevented, was quickly and rudely exposed in the progress of the case.

#### CASE XIX.—*Rheumatic Endo-pericarditis; Recovery.*

Matthew R., aged thirty-three years, carpenter, intemperate, admitted March 11th, 1872, for subacute rheumatism engaging the smaller joints of the feet and hands, and migratory in character. He was thin, pale, and weak, suffering much from articular pain, which deprived him of sleep, but was not accompanied by swelling or cardiac complication. The treatment consisted in large doses (fifteen and thirty grains respectively) of bicarbonate and acetate of potash, with five drops of tincture of aconite, repeated every second hour: it was not, however, attended with much benefit. Opium was subsequently given in gr. j doses every fourth hour, with decided relief of pain; and the affected

joints were stupefied with warm laudanum, and wrapped in carded cotton.

March 25th. Pulse 96, soft, and regular; some blood in sputa; base of left lung congested, and loud sawing *frottement* audible all over precordium and left front; also towards the right side, as far as the right sterno-clavicular articulation, but not in the neck or posteriorly; it is double, corresponding to the sounds of the heart, but audible during expiration only; no *frémissement*, or carotid pulsation; stabbing pain at the apex of the heart; breathing tranquil, and 24 in the minute. Precordial pain was entirely relieved by the application of two leeches, followed by a warm poultice. Calomel was given in doses of gr. j every hour. On the following day a mitral systolic and substitutive murmur existed at the apex; and on the 27th, a single friction-sound was audible at the left base, and at the apex a double *frottement*, through which the soft systolic mitral murmur was distinguishable. Pulse 108. No increase of precordial dullness. This man was discharged in the first week of April quite free from pain. No precordial friction-sound then existed, but the apex-systolic murmur was still distinctly audible.

CASE XX.—*Strain, followed by Pain in the chest, Dyspnœa, and Dysphagia; Dulness on Percussion over the left side of the chest with absence of Respiratory sound and of Vocal Frémitus; Displacement of the Heart to right side; Pericardial Frottement; Remarkable Rapidity of Pulse; Aggravated Dyspnœa, and Orthopnœa; Death by Asthenia. Copious Effusion of Serum into the left pleural cavity; Cancerous Solidification of the left lung, with Adhesion to the Pericardium and chest-wall, and remarkable Cancerous Thickening of the left pleura; Cancerous Transformation of the base of the right lung and right pleura in an earlier stage, with Adhesion of the Lung to the chest-wall; Cancerous Pericarditis, with Partial Adhesion to the Heart, which was soft, but not otherwise diseased; Cancer of the liver, mesentery, omenta, and left supra-renal capsule.*

Michael R., aged twenty-two, a labourer, was admitted into the Mater Misericordiæ Hospital on the 2nd of January, 1866. He

had enjoyed general good health up to July of the preceding year, when, on pushing from him with both his hands a large clamp of turf, he experienced in the region of the heart a sensation as if something had burst, followed by a feeling of scalding and tightness, and by shortness of breath; his head "swam," his sight became dim, and he was obliged to sit down. After a few hours he recovered sufficiently to be able to go home. This feeling in the region of the heart continued some days; it then passed away, and his breathing was so much improved that he was able to resume his work. He did not, however, feel quite well.

About three months subsequently he felt some difficulty in swallowing. The morsel in its descent was stopped behind the lower end of the sternum and returned some distance, but after an effort it passed onwards into the stomach. After swallowing a few mouthfuls in this way the food passed with less difficulty. Liquids passed with facility, but he could not swallow solids without taking a mouthful of drink.

About this time, also, he caught cold whilst attending his mother in her last illness, and had cough, muscular pains in the neck and back, and stitch in the left side. Whilst journeying to town on the preceding day he became jaundiced in the train. When admitted, he was of a deep lemon-colour, and the urine was loaded with bile-pigment. Dysphagia as above described. Pulse 108, and feeble, but regular; respiration 36; tongue slightly coated; orthopnoea; liver prominent, especially the left lobe, tender to pressure, and projecting two inches below the costal cartilages; bowels constipated. Left side of chest universally and absolutely dull, and on this side no respiratory sound audible except posteriorly, above the angle of the scapula, where it was faintly bronchial, and where likewise the voice was ægophonic; vocal vibration was abolished on this side, which in girth exceeded the right by half an inch, was smooth by obliteration of the intercostal spaces, and motionless during respiration. Behind the anterior fold of the axilla some distended superficial veins were visible. The heart pulsated strongly behind the right margin and lower portion of sternum; its sounds were clear and unassociated with murmur. Preceding the first sound, however, an obscure grating, regarded as pericardial, was

audible. On the following day this grating sound was somewhat "to-and-fro" in character. The pulsations of the heart were heard all over the chest, but were especially loud over the left side, and were transmitted, but not with intensity, in the course of the aorta. Over the right side resonance was clear, and respiration loud.

Six leeches were applied to the epigastrium, followed by a poultice; and two grains of mercury with chalk, and an equal quantity of James' powder, were given every third hour.

January 6th. Pulse 162, weak, and intermitting; respiration 42.

7th. Cannot lie down; dyspnœa very urgent; takes rest by leaning over on a pillow placed upon a table to his left; no lividity of surface; crepitus heard in right mammary region; respiration and resonance elsewhere normal on that side; pericardial *frottement* no longer audible. To have a terebinthinate enema, and, every fourth hour, a draught composed of sulphuric ether, 3ss, and sedative liquor of opium, ℞ xv.

8th. Dulness over base of right lung posteriorly, and friction in mammary region, where crepitus had been previously heard. Up to this date the diagnosis was by no means clear. The history of the case, and the existence of dysphagia were suggestive of aneurism; but there were obvious and insuperable objections to this view.

9th. Pulse 174; respiration 42; great respiratory distress, and fulness with pain in epigastrium.

13th. Has improved under the use of mercurial inunction, aperient enemata, blisters applied over the chest and epigastrium, and repetition of anodyne draughts. Can now swallow solids in small morsels. Heart's sounds most distinctly audible over midsternum, where a soft bellows-murmur preceded the first sound, but fell short of it. No lividity or œdema; jaundice continued.

15th. Is sinking. Voice nasal, and diphtheritic deposit on inside of cheeks. Died at 8 P.M.

Body examined fourteen hours after death; was rigid, and not much emaciated.

*Abdomen.* Peritoneum greatly thickened, and opaque; liver



much enlarged, olive-coloured, and exhibiting several large cancerous nodules upon its surface, and throughout its substance; gall-bladder large and distended with bile; pancreas had been converted into a large nodulated scirrhus mass, involving the common biliary duct. The mesentery and omenta were pervaded by cancerous nodules, varying in size from that of duck-shot to that of a musket-ball. Right supra-renal capsule attached to liver, and in a state of cancerous transformation. Left capsule, both kidneys, spleen, and hollow viscera, healthy.

*Chest.* Left pleural cavity full of straw-coloured serum, and pleura, both parietal and pulmonary, greatly thickened and roughened by cancerous nodules and effused lymph, the latter quite as thick as upper-leather. Both lungs were attached to the walls of the chest by adhesions which, posteriorly, were remarkably strong. The anterior edge of the left lung was incorporated with the pericardium by thickened pleura, which here was as thick as sole-leather. On section, the lung was found to have been converted into a mass of scirrhus; it was solid, rigid, and heavy. The base of the right lung was similarly altered, but in an earlier stage; the pulmonary pleura was thickened, and from its deep surface thick bands passed into the substance of the lung, branching, and dividing the surface of section into polygonal areolæ, in which healthy and crepitant lung tissue was lodged. The apex of the right lung was apparently sound, and was resonant and crepitant. The surface of the heart was rough and granulated; it was of a dark red colour, and attached to the pericardium by reticulated bands of adhesion, which were easily broken down, and obviously recent. The heart was soft, and the valves were all in a healthy state.

## CHAPTER VI.

### DISEASES OF THE SUBSTANCE OF THE HEART.

DISEASES of the substance of the heart may be conveniently divided into :

1. Morbid changes of volume.
  - (a) Hypertrophy.
  - (b) Atrophy.
  - (c) Dilatation.
  - (d) Aneurism.
2. Changes in consistence.
  - (a) Softening (inflammatory, typhoid, fatty).
  - (b) Induration.
3. Adventitious products.
  - (a) Adipose (fatty accumulation, fatty substitution, fatty transformation).
  - (b) Hydatids.
  - (c) Fibroid growth (hyperplasia).
  - (d) Osteoid growth.
  - (e) Tubercle.
  - (f) Cancer (medullary, scirrhus, melanotic).
  - (g) Syphiloma.
4. Wounds of the heart.
5. Phlegmasiæ.
  - (a) Myocarditis.

*Hypertrophy* of the heart is due essentially to an effort of compensation. The heart being in a state of healthy nutrition, it suffers, owing to one or more of the causes to be presently enumerated, from a derangement of the normal equilibrium between its dynamic capacity, as the central organ of the circulation, and the conducting properties of the vascular system.

Bertin describes hypertrophy and atrophy as the result of an "augmentation" and a "diminution" of nutrition, respectively ;

whilst induration and softening he regards as "alterations" of nutrition.\*

Lebert defines hypertrophy, in general, as "an augmentation of the essential constituent molecules of a tissue or organ, a veritable increase of nutrition, with excess of its materials."† As regards the heart, Rindfleisch understands hypertrophy to be "an increase of the volume of the heart, which has its foundation in a hyperplasia of the myocardium."‡

According to the last named writer, therefore, hypertrophy of the heart has always an inflammatory, and therefore a morbid origin. This doctrine may, however, be questioned on two grounds; namely, the presence, by universal admission, of the products of inflammation in hypertrophied hearts only exceptionally; and the strict proportion observed, as in all other examples of muscular hypertrophy, between the increase of development of the heart and the augmentation of its function, in every case, without exception, of hypertrophy of that organ.

Hypertrophy of the heart may be either general or partial; and, being the result of an effort of compensation, it implies either that the portion affected, if in a healthy state, has undergone simple increase in the thickness of its walls, proportionate to the difficulty imposed upon it in carrying on the circulation; or that, being incapacitated for such increase beyond a certain limit, by malnutrition, or degenerative change, it has yielded to the obstacle opposed to it, and become also dilated.

Hence there can be only two forms of hypertrophy; namely, the *simple* form, in which the walls are thickened without alteration in the size of the cavity; and the *excentric* form, in which the walls are thickened and the cavity dilated.

To these two varieties of hypertrophy, Bertin§ has added a third, which he names *concentric*. In this, the walls are thickened, and the cavity is reduced in capacity. Hope recognizes,||

\* *Maladies du Cœur*, p. 345.

† *Traité d'Anatomie Pathologique*, 1857, tom. i., p. 85.

‡ *Text-Book of Pathological Anatomy*, 1872, p. 226.

§ *Mem. de l'Académie Royale des Sciences*, 1811; *Traité des Maladies du Cœur*, 1824, p. 283.

|| *The Diseases of the Heart and the Great Vessels*, third edition, 1839, p. 234.

as the equivalents of the foregoing, three forms of hypertrophy, namely :

1. Simple hypertrophy.
2. Hypertrophy with dilatation (active aneurism of Corvisart, excentric or aneurismal hypertrophy of Bertin).
3. Hypertrophy with contraction (concentric hypertrophy of Bertin).

Of the second or *excentric* variety, two forms have been recognized ; namely (*a*) that in which the walls are thickened and the cavity dilated ; and (*b*) that in which the cavity is dilated, but the walls retain their natural thickness.

This latter form Hope recognizes under the designation of "hypertrophy by increased extent of the walls;" and Latham by the more simple and appropriate title of "proportionate"\* hypertrophy. But inasmuch as the state of hypertrophy is constituted essentially by thickening of the walls, I cannot admit this sub-variety, in which that condition is absent. I prefer including it in the category of dilatations, under the title of the *simple* form of that condition of the heart. The states of hypertrophy and dilatation being relative, must be judged by reference to a fixed standard of comparison.

Laennec roughly estimated the volume of the heart, the thickness of its walls, and the capacity of its chambers, absolutely and relatively, as follows:—"The heart, comprising the auricles, ought to have a size equal to, a little less, or a very little larger than, the fist of the subject. The walls of the left ventricle ought to have a thickness a little more than double that of the walls of the right ; they ought not to collapse when an incision is made into the cavity. The right ventricle, a little larger than the left, and having larger columnæ carneæ notwithstanding the inferior thickness of its walls, ought to collapse when an incision has been made into it."†

Bouillaud's measurements, though admittedly based on insufficient data, may be regarded as approximately correct.‡

He estimated the volume, weight, and thickness of the heart

\* *Lectures, etc., comprising Diseases of the Heart*, 1846, vol. ii., p. 182.

† *Opus citat.*, vol. ii., p. 404.

‡ *Vide antea*, p. 40, for Table of Comparative Measurements, etc., relating to normal heart.



in the young and in the adult subject respectively under three heads,\* viz. :

1. Normal heart.
2. Atrophied heart.
3. Hypertrophied heart.

#### Of the normal heart

				Ozs.	Drms.
The maximum weight is	...	...	...	11	0
The minimum „	...	...	...	6	2
The mean „	...	...	...	8	3

The average weight of the heart in males, between the ages of twenty-five and sixty years, he estimates at eight to nine ounces; and between sixteen and twenty-five years, one to two ounces less. In persons very large and strong, it might amount to eleven ounces without transgressing the limits of health. In females, the weight of the heart is less than the above by an undetermined quantity.

Circumference of the normal heart at the base of the ventricles :

					Inches.	Lines.
Maximum	...	...	...	...	10	6
Minimum	...	...	...	...	8	0
Mean ...	...	...	...	...	8	9

#### Length from root of aorta to apex :

Maximum	...	...	...	...	4	0
Minimum	...	...	...	...	3	2½
Mean ...	...	...	...	...	3	7⅓

#### Transverse diameter a little below base of ventricles :

Maximum	...	...	...	...	4	6
Minimum	...	...	...	...	3	5
Mean ...	...	...	...	...	3	7½

#### Thickness of walls of left ventricle at base :

Maximum	...	...	...	...	0	8
Minimum	...	...	...	...	0	5
Mean	...	...	...	...	0	6

#### Right ventricle at base :

Maximum	...	...	...	...	0	3½
Minimum	...	...	...	...	0	1½ to 2
Mean ...	...	...	...	...	0	2⅓

\* *Traité Clinique des Maladies du Cœur*, 1835, tom. i., Prolégomènes, p. 3-4.

A general estimate of the thickness of the ventricular walls would yield,

					Lines.
For the left ventricle	...	...	...	...	7
For the right ventricle	...	...	...	...	2½

From sixteen to forty years, the thickness increases, being greater in persons of strong build and tall, than in those of opposite physique.

Thickness of interventricular septum is 11 lines.

Thickness of walls of left auricle :

					Lines.
Maximum	...	...	...	...	2
Minimum	...	...	...	...	¾ to 1
Mean	...	...	...	...	1½

Thickness of right auricle :

Maximum	...	...	...	...	1½
Minimum	...	...	...	...	½
Mean	...	...	...	...	1

As to capacity, each of the ventricles would contain a pullet's egg, but the right somewhat exceeds the left in size. The auricles are equal in capacity respectively to the corresponding ventricles, the right somewhat exceeding the left.

Clendinning, as quoted by Hope,\* gives the following as the average weight of the heart in the male and female respectively, from fifteen years upwards :

			Males.	Females.
15 to 30 years	...	...	8¼ ozs.	8½ ozs.
30 to 50 „	...	...	8½ „	8½ „
50 to 70 „	...	...	9½ „	8 „
70 and upwards	...	...	9¾ „	8 „

		Atrophy.	Normal State.
Maximum weight	...	200 grammes	350 grammes.
Minimum	„	135	200
Mean	„	175	262

\* *Opus citat.*, p. 237.

		Hypertrophy.	Normal state.
Maximum weight of heart	...	688 grammes	350 grammes.
Minimum	„	338 „	200 „
Mean	„	473½ „	262 „

Thus, the heaviest heart which Bouillaud has met with weighed only twenty-one and a-half ounces.

Lobstein has seen one of thirty-two ounces.

The heaviest heart which I have met with in my own practice weighed only thirty-two and a-half ounces ; but I have seen and preserved one obtained from a patient of my colleague, Dr. John Hughes, which weighed forty-two ounces.

Hope mentions one which he had seen of forty-four ounces in weight.\*

Doctor Stokes, as already mentioned, exhibited at the Pathological Society a heart which had been the subject of valvular disease and adhesion of the pericardium, yielding the enormous weight of four pounds and two ounces, avoirdupoise. Allan Burns adduces the authority of Lieutaud for the description of a heart which weighed five pounds ; and he mentions an example of simple hypertrophy of “several pounds weight,” which he had himself seen.†

Thus, it is manifest that the maximum weight of an hypertrophied heart witnessed by Bouillaud has been greatly exceeded. Nor can I admit his assertion that a heart of eleven ounces, although close on the confines, is not within the limits of morbid growth. I am willing to believe that in a strong man of very active habits, a heart of eleven ounces may consist with a state of apparent health, the enjoyment of society, and even a continuance, without abatement, of active duties and indulgences. But, in such a case, an attack of serious illness of any kind, especially one of an acute febrile character, will show, by the tumultuous action of the heart, and by its early failure, that the condition of that organ, antecedent to present illness, was not that of health.

\* *A Treatise on the Diseases of the Heart and Great Vessels*, 1839, p. 237.

† *Observations on some of the Most Frequent and Important Diseases of the Heart*, etc., 1809, p. 40.

Hypertrophy of the chambers of the heart may be *primary* or *consecutive*, and due to :

1. Mechanical obstruction to the circulation, arising from
  - (a) Obstruction at valvular orifice in front.
  - (b) Incompetence of valves in front.
  - (c) Incompetence of valves behind (mitral).
  - (d) Rigidity of main arteries.
  - (e) Spasmodic resistance of small arteries.
2. Habitual over-exertion of the voluntary muscular system.
3. Habitual nervous excitement of the heart.

The two last mentioned causes (Nos. 2 and 3) are alone capable of producing primary or uncomplicated hypertrophy of the heart; but, inasmuch as hypertrophy without complication, causative of, or dependent upon it, is never fatal, the opportunity for examining a heart affected with this form of hypertrophy, unassociated with antecedent or consecutive disease, is presented only where death in such a case is caused by acute febrile disease, or by accident.

The changes consecutive to hypertrophy of the heart, and in the order of their frequency, are,

1. Tissue degeneration of the heart, granular and fatty.
2. Dilatation of either ventricle, or of both ventricles.
3. Atheroma of the aorta and its branches.
4. Dilatation of the aorta, and consequent inadequacy of the aortic valves.
5. Fatty degeneration of the kidneys.
6. Calcareous transformation of the aortic valves.

Hypertrophy of the heart from severe and long-continued mechanical labour is the result of a compensatory effort on the part of that organ; additional duty has been imposed upon it; its rate and force of contraction have been proportionately increased, and so long as its nutrition may be quickened in an equal ratio, so long will increase in the thickness of its walls keep pace with, and strictly counterbalance exaggerated function. Ultimately, however, owing to impairment of primary digestion, or of assimilation, nutrition fails, or rather, it is no longer capable of being maintained at this high standard; degenerative changes in the tissue of the heart are now set up; its



power of resistance to excentric pressure is proportionately lessened, and dilatation follows. Concurrently with degeneration of the heart, but oftener consecutively to it, atheromatous change in the coats of the aorta and smaller arteries takes place, and the kidneys undergo the fatty change of structure. The period occupied by these successive processes may be very considerable, varying from six months to as many years; and should the case in its complex form be now for the first time presented to a physician, it would be difficult indeed to determine with certainty the order of sequence of the several changes indicated.

I have no doubt whatever that hypertrophy of the heart, arising primarily from excessive muscular exertion, or from habitual emotional excitement, whether of joy, fear, anxiety, or anger, but ultimately associated with renal disease by general progressive degeneration of tissue, has been repeatedly set down as a consequence of the renal affection.

Doctor Quain\* classifies all cases of hypertrophy of the heart under three heads, according to the tissue primarily and principally involved; namely, hypertrophy of,

1. The muscular tissue.
2. The connective tissue.
3. The adipose tissue.

The *causes* of hypertrophy he regards as of three kinds; viz.,

1. Nervous.
2. Mechanical.
3. Nutritive.

The first is exemplified in the primary enlargement so common in periods of popular excitement. The second class of causes may be located in the heart itself, in the great vessels, or in the small and distant vessels, as exemplified in Bright's disease, and in pregnancy.

The causes which operate through nutrition may be general (anæmic) or local (inflammatory); they are exemplified respectively in the enlargement of anæmia, which, however, is usually the result of dilatation rather than hypertrophy; and in that consecutive to pericarditis, and to inflammation of the connective tissue of the heart. In both these latter instances the

\* Lumleian Lectures, *Medical Times and Gazette*, March 23rd, 1872.

enlargement is due to hypertrophy of the interstitial fibrous tissue of the heart, and constitutes a veritable hyperplasia; it is remarkably prone to changes of degeneration.

In connective tissue hypertrophy, Quain remarks, the walls are dense and leathery, and the colour of various tints, from that of muscular fibre to a light grey hue.

Under the microscope fibrous tissue is seen in all stages of development between, and pressing upon, the muscular fibre.

Corvisart long since described this form of hypertrophy of the heart, and attributed it to inflammation of the connective tissue of the organ.\*

Rokitansky even doubts the increase of muscular substance in any form of hypertrophy, and intimates that, in his opinion, the increased bulk is due in all cases to excessive development of the areolar tissue of the organ.†

Laennec, Sir W. Jenner, Williams, and Ormerod, have likewise recognized hypertrophy of the areolar tissue of the heart.

Nevertheless, I doubt that it constitutes a form of hypertrophy involving increased volume of the heart. Hypertrophy of the connective tissue of other organs, the liver, the spleen, and the lungs, leads to atrophy and general diminution of volume, not to hypertrophy of their parenchyma. I have not met with an example of hypertrophy of the heart, excepting those in which its bulk was increased from superficial deposit of fibrin, the result of repeated accessions of pericarditis, in which increased development of connective tissue was the cause of augmentation of its volume. It is theoretically conceivable that plastic exudation, following close upon inflammatory irritation of the fibrous tissue of the heart, might give rise to enlargement of the whole organ. But even in passive or glandular organs, as is well known, such effect, though actually produced, is only temporary, atrophy and induration being consecutive and proximate changes. In the heart, subject as that organ is to the operation of high pressure from within, repeated fifty to eighty times or oftener every minute, and tending to stretch its walls and di-

\* *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels* translated by Hebb, p. 56.

† *Pathological Anatomy*, Sydenham Society's edition, vol. i., p. 40.

late its cavities, hyperplastic increase of the fibrous element, by lessening the resisting power of its walls to internal pressure, determines, even more rapidly than in other organs, atrophy of substance, and dilatation of the parenchymatous recesses or cavities of the organ.

Rindfleisch even maintains that inflammation of the connective tissue of the heart, by extension from the endocardium, and limited to a small space, is the primary cause of partial aneurism of the heart, by determining thinning of its walls.

Fatty hypertrophy differs from fatty degeneration, as consisting in accumulation of fat beneath the pericardium and between the muscular fibres.

As *causes* of hypertrophy, Corvisart mentions all such as give rise to obstructed circulation; moral causes; severe labour or exercise; and, "perhaps," the stimulant quality of the blood.

To the preceding causes Bouillaud adds chronic inflammation of the pericardium or endocardium.

Arising from the former causes, hypertrophy would be "primitive," according to his nomenclature; and arising from the latter it would be "consecutive."

Hope, Lebert, Quain, and indeed most modern authorities, likewise hold that inflammation is a direct and common cause of hypertrophy.

"Concentric hypertrophy," as first noticed and described by Bertin, has been likewise admitted by Bouillaud, who gives three cases in illustration,\* by Laennec,† Hope,‡ Rokitansky,§ and Lebert.||

For the reasons previously stated, I venture to question its occurrence as a distinct pathological change; and further, because I have not met with a single example of the condition of the heart corresponding to the "concentric hypertrophy" of Bertin, that would not admit of the explanation of Cruveillier previously referred to, namely, thickening of the walls and reduction of the cavities of a heart in a state of simple hypertrophy, by spasmodic contraction at the moment of sudden

\* *Opus citat.*, tom. ii., observations 118, 119, 120.

† *Traité d'Auscultation Mediate*, 1826, tom. ii., p. 412.

‡ *Opus citat.*, p. 234.

§ *Pathological Anatomy*, vol. i., p. 156.

|| *Traité d'Anatomie Pathologique*, 1857, tom. i., p. 85.

death; or which was not an example of atrophy with contraction of the cavities.\* In this opinion I am fortified by the observations of Dr. George Budd, who states that of eight cases collected by him, exhibiting the condition of heart regarded as peculiar to concentric hypertrophy, not one presented signs during life, or *post mortem* evidence of obstructed circulation through the heart. There was no irregularity of pulse, no dropsical effusion, and after death the right chambers of the heart were found not dilated. He adds, with much force, that it is impossible to conceive that a left ventricle, not capable of containing an almond, should present no obstacle to the circulation during life.†

In Laennec's case the walls of the left ventricle were one and a-half inch thick, and its cavity not large enough to contain an almond stript of its shell; yet, the day preceding death the breathing was free, and "nothing (he says) led me to suppose that this man had a disease of the heart."

Bertin gives the particulars of four cases of concentric hypertrophy.‡ In the first the pulse was regular, small, hard, and strong; the pulsations of the heart strong and concentrated, and the sounds dull and seemingly remote. On examination of the body the walls of the left ventricle were found an inch thick in the middle, the fleshy columns thickened, the cavity much diminished, the aorta reduced in calibre, and scaly, and the other chambers and valves sound. There was softening of the brain, and extensive disease of the arteries generally.

In his second case, there had been great embarrassment of breathing. The right chambers were thin and dilated, the left auricle normal, the wall of left ventricle one and a-half inch thick near the base, and its cavity reduced to half its normal capacity; the aorta was atheromatous.

In his third case there was likewise great oppression of breathing. On examination after death the left ventricle was found double its natural thickness, and its cavity reduced to the size of an ordinary filbert. The other cavities, and all the valves, were in a healthy condition.

\* *Dictionnaire de Médecine et Chirurgie Pratique*, Article "Hypertrophie."

† *Medico-Chirurgical Transactions*, vol. xxi., 1838.

‡ *Opus citat.*, p. 310, observations 80, 81, 82, 87.



The fourth case was an example of concentric hypertrophy affecting the right ventricle only. Its walls were eleven to sixteen lines thick, and its cavity reduced to the size of a pigeon's egg. The foramen ovale was patent, and the pulmonary artery nearly closed by a diaphragm.

In the first of the cases just given in summary, the diseased state of the aorta and of the arteries generally, was sufficient to account for hypertrophy of the left ventricle, which was probably in the simple form during life; a view seemingly confirmed by the character of the pulse, and of the heart's action.

In the second case, the atheromatous condition of the aorta would imply a state of hypertrophy, simple or dilated, of the left ventricle, and the dyspnoea would be, to my view, satisfactorily accounted for by the atheromatous state of the aorta, and the attenuation of the right ventricle. In the third case details are wanting, *e.g.*, as to the period during which dyspnoea had existed. Otherwise this would seem the only one of Bertin's cases which might be adduced with effect, in support of his doctrine of the existence of concentric hypertrophy as a primary pathological alteration; as the other cavities and all the valves of the heart were sound.

Bertin explains hypertrophy of the right ventricle in his fourth case by reference to the entrance of arterial blood through the foramen ovale, from the left to the right auricle, and consequent "nutritive irritation" of the walls of the right ventricle.

But it may be remarked that such cause should give rise likewise, and in the first instance, to hypertrophy of the right auricle, which, however, did not exist. Theoretically, it would seem that for the production of a state of concentric hypertrophy of the right ventricle three conditions should coexist; namely, a direct passage of blood from right to left auricle, through an open foramen ovale; competency of the tricuspid valve; and contracted pulmonary orifice, by which an obstacle is opposed to the escape of blood from the right ventricle.\* The authority of Bertin is entitled to so much respect in con-

\* An example of contracted pulmonary artery is given by Morgagni, *De Sedibus et Causis Morborum*, tom. ii., epistol. xvii., p. 525.

nexion with any subject of cardiac pathology, that I have felt it necessary to explain at length my reasons for dissenting from him on this subject.

There is much disagreement amongst pathologists as to the most common form of hypertrophy. Corvisart considers that with dilatation or "active aneurism," the usual form.

Rokitansky declares that simple hypertrophy is rarely met with, and oftener in the left than in the right ventricle; it is, according to him, a transitional state, and merges into the excentric form, which is, *par excellence*, the typical form of hypertrophy. The side affected is most frequently the left, whence the pathological change extends to the right, and when both are affected, the condition constituting *cor taurinum* is established. When the auricles are affected with hypertrophy, the change is due to contraction of the corresponding auriculo-ventricular orifice.\*

I am of opinion that hypertrophy is, in the great majority of instances, associated with some degree of dilatation.

In this opinion I am sustained by the recent authority of Rindfleisch, who goes the length of declaring that "a certain degree of dilatation is associated with every hypertrophy of the heart."† Whilst maintaining that hypertrophy of the heart is most frequently presented under the excentric form, I cannot subscribe the exclusive doctrine of Rindfleisch. Simple hypertrophy is certainly, and not unfrequently, met with; but always, as Rokitansky asserts, as a state of transition from health to dilated hypertrophy. It is, *par excellence*, the condition in which the left ventricle is found in the early stage of hypertrophy from renal disease; the stage, namely, of considerable duration, which precedes nutritive decay and tissue degeneration of the organ. When these changes have set in, as they invariably do in the ordinary course of pathological sequence, the ventricle undergoes dilatation, usually in excess of antecedent hypertrophy, and excentric hypertrophy of a degenerated left ventricle is the result. But, inasmuch as the impairment of general nutri-

\* *Opus citat.*, vol. ii., p. 156-7.

† *Text-Book of Pathological Anatomy*, translated by Kloman and Miles, 1872, p. 227.

tion causative of, or attendant upon renal disease, must operate upon the entire heart, the right ventricle likewise suffers; but not having previously undergone hypertrophy, the condition in which it is found is usually that of dilatation with thinning of the walls, and granular or fatty degeneration of the muscular fibre.

Walshe states that general hypertrophy of the heart is always of the excentric kind, and that the individual chambers are affected in the following order of frequency: viz., left ventricle, left auricle, right ventricle, and *longo intervallo*, right auricle.\*

Hypertrophy, according to Stokes, is rarely confined to one cavity.† But hypertrophy of the left ventricle of renal origin, where the valves are free from disease, constitutes an exception to this statement. The other chambers undergo no change till tissue-decay and dilatation of the left ventricle have set in, as they do in ordinary pathological succession; and then, participating in the effects of malnutrition, and influenced by the progressive incapacity of the left ventricle in front, they undergo gradual dilatation of the atrophic or attenuative kind.

Mechanical obstruction to the circulation, of whatever kind, was the only cause of hypertrophy ("active aneurism") recognized by Corvisart. For the right auricle, the obstacle must be located at the tricuspid orifice; and for the right ventricle, at the orifice of the pulmonary artery, or in the lungs. For the left auricle the cause of obstruction must be located at the mitral orifice; and for the left ventricle it may consist in valvular obstruction at the mouth of the aorta, tortuosity of the arteries, dropsical effusion, or capillary obstruction by spasm of the skin.‡

The last mentioned cause is of special interest to modern pathologists, as indicating on the part of Corvisart, a knowledge of the connexion, at least in their remote relationship, between renal disease and hypertrophy of the left ventricle of the heart. A rigid, pallid, unperspiring state of the skin, constitutes one of the earliest and most suggestive of the general symptoms of

\* *Opus antea citat.*, p. 277-8.

† *The Diseases of the Heart and Aorta*, p. 273.

‡ *On the Diseases and Organic Lesions of the Heart and Great Vessels*, translated by Hebb, p. 107.

renal disease. The cause of this state of the integument was unknown to Corvisart; yet the condition itself, and its frequent association with tumultuous action and enlargement of the heart, did not escape his observation. "Active aneurism," according to him, is more common in the left ventricle than in the right, because the former is more irritable, and under a stimulus, more susceptible of active nutrition; and finally, because obstacles to the circulation are of more frequent occurrence at the orifice of the aorta than at that of the pulmonary artery.\* Causes which give rise to "active aneurism" of the left ventricle, usually produce, consecutively, "passive aneurism" of the right.

Singularly enough, whilst asserting that mechanical obstruction is the only cause of hypertrophy, he propounds the paradox, that in that condition of the heart the increased power is in excess of the resistance to be overcome.

Bouillaud,† in addition to the foregoing, recognizes moral agency in the production of hypertrophy. He likewise mentions as causes, severe and long continued bodily labour or exercise, and chronic inflammation of the pericardium and endocardium. As already stated, he designates hypertrophy arising from the last mentioned cause as "consecutive," and that produced by all those previously mentioned, as "primitive." He gives thirty-three cases of chronic peri- or endocarditis, in all of which hypertrophy existed; but he does not inform us whether valvular disease was likewise present in these cases; an omission which renders his premises of little value in regard to the conclusion he desires to draw from them. Amongst the possible causes of hypertrophy of the right ventricle, he includes the entrance of blood from the left ventricle, through a patent foramen ovale, or perforated ventricular septum, on the principle of "excitation," and gives the particulars of four cases,‡ in each of which the twofold lesion existed. The pathogenic connexion, however, he considers as "somewhat hypothetical."

Lebert mentions, amongst the remote causes of hypertrophy of the heart, the tubercular diathesis, and gives a case in illus-

\* *Opus citat.*, p. 66.

† *Traité Clinique des Maladies du Cœur*, 1835, tom. ii., p. 365, et suivant.

‡ Observations 75, 76, 77, and 79.



tration, in which, however, tubercular pericarditis had been set up.\*

Of one hundred and seventy-one cases of phthisis collected by Quain, the heart was enlarged in 25·66 per cent. in males, and only in 7·0 in females. In males the heart was small in 53 per cent., and in females in 67 per cent. In males it was normal in 21 per cent., and in females in 26 per cent. In a girl of eight years, the heart weighed only two ounces; and in another, aged fourteen years, it weighed only one ounce and fourteen drachms.† It is not stated in what proportion, if any, of the cases in which hypertrophy existed, other causes of enlargement were present.

I have not seen hypertrophy of the heart in bodies exhibiting evidence of extensive tubercular development, or advanced pulmonary phthisis, in which one or more of the ordinary causes of hypertrophy did not likewise exist. These causes, too, renal or cardiac, judging from the stage to which they had attained, as well as from the history of the patients' last illness, were anterior in date to the phthisical affection.

Hermann Lebert has recently expressed the opinion, in support of which he gives several illustrative cases, that in relation to pulmonary tuberculosis, diseases of the heart and aorta, such as are capable of giving rise to impairment of general or special nutrition, stand in the position of cause, not effect.‡

Rokitansky goes the length of declaring hypertrophy and dilatation of the heart to be incompatible with tuberculosis; atrophy being the usual condition of the heart met with in that state.§ He justly attributes hypertrophy with dilatation of the right chambers in many instances to curvature of the spine and pulmonary emphysema,|| and mentions, as producing a similar effect, narrowing of the mitral orifice.

Obstruction is, *par excellence*, the cause of hypertrophy in the

\* *Traité d'Anatomie Pathologique*, 1857, tom. i., observation clxiv., p. 555.

† *Lcco citat.*

‡ *Medical Times and Gazette*, December 11th, 1869.

§ *Pathological Anatomy*, vol. i., p. 165 and 316.

|| *Opus citat.*, vol. i. p. 163-4.

opinion of Hope, whilst constant congestion of the cavities is, in an equal degree, the cause of dilatation.\*

Walshe classifies the causes of hypertrophy under seven heads; viz.,†

1. Causes originating in the system.

(a) Excessive nourishment, especially nitrogenized, with stimulants and sedentary habits.

(b) Excessive exercise, *e.g.*, walking, rowing.

2. Causes originating in the blood.

(a) Uræmia (?).

(b) Rheumatic hyperinosis (?).

3. Causes originating in the heart itself.

(a) Functional excitement, as in prolonged anæmia, and chronic pericarditis.

(b) Mechanical obstruction; (affecting left ventricle), aortic constriction, aortic regurgitation, mitral regurgitation, mitral constriction (?), mitral regurgitation and constriction combined; (affecting right ventricle), tricuspid regurgitation, pulmonary constriction, mitral regurgitation through engorgement of lungs; (affecting left auricle), mitral regurgitation and constriction, aortic regurgitation and constriction, but in much less degree; (affecting right auricle), tricuspid regurgitation.

4. Causes originating in the great vessels.

(a) Pressure obstructing their interior.

(b) Smallness or constriction of aorta.

(c) Aneurism of aortic arch near the heart (?).

(d) Diminished elasticity of the coats of the aorta or pulmonary artery, affecting left or right ventricle.

5. Causes originating in the lung-circulation, and affecting right ventricle.

(a) Chronic bronchitis.

(b) Emphysema.

(c) Contraction after pleurisy.

(d) Dilatation of bronchi and cirrhosis of lung.

\* *A Treatise on the Diseases of the Heart and Great Vessels*, 1839, p. 246.

† *The Diseases of the Heart*, 1863, p. 280. The notes of interrogation are in the original.

(e) Diminution of cavity of chest by deformity or by abdominal tumors (?).

6. Causes originating in the kidneys.

(a) Persistent obstruction in their capillary circulation (?).

7. Causes originating in systemic capillaries.

(a) Prolonged obstruction of any given mechanism in some large portion of the systemic capillaries (?).

He doubts Bright's theory of hypertrophy in renal disease, regarding it as like the renal affection itself, "an additional local expression of the main diathesis," and as in part arising likewise from specific irritation of the blood on the endocardium.\*

Doctor Stokes, whilst admitting hypertrophy from other causes, declares that in the absence of disease in the valves or aorta, it is exceptional.† But in the various forms of Bright's disease, now so commonly met with in our hospitals, hypertrophy of the heart, confined to the left ventricle, is rather the rule than the exception.

I cannot admit a special form of hypertrophy under the designation of "fatty," as mentioned by Quain, who describes the fat as mixed up with the muscular fibres throughout, but in less proportion towards the interior, and immediately beneath the endocardium limited to a few fat cells at intervals between the fibres.‡ Fat thus deposited among the muscular fibres of an hypertrophied heart, should be regarded as the result of consecutive and degenerative, not primary or constructive changes. The only example of fatty hypertrophy which can be admitted is of a *quasi* character, namely, that of a heart magnified by deposition of fat upon the outer surface, but in reality atrophied *quoad* its muscular substance (*vide* "Aneurism," case of Mr. B.).

Hypertrophy of the heart has been attributed to pregnancy by some writers. Thus, in 1828, Lacher announced, as the result of his examination of one hundred and thirty healthy women who had died in child-birth, that the heart was hypertrophied in all; the left ventricle, which was the part affected, being increased in thickness from a quarter to half an inch.§

\* *Opus citat.*, p. 284.

† *The Diseases of the Heart and the Aorta*, 1854, p. 345.

‡ Lumleian Lectures, *Lancet*, March 23rd, 1872.

§ Doctor Quain's Lectures, *loco citat.*

This form of hypertrophy, which has been attributed to the pressure of the gravid uterus upon the abdominal arteries, and to the increased circulation in the uterus itself, must, like the growth of the latter organ at that period, be of only temporary duration, and subject, like it, to consecutive involution. Hypertrophy of the left ventricle in connexion with albuminuria was first explained in 1817 by the late Mr. James of Exeter, as caused by obstruction in the systemic capillaries,\* and subsequently by Bright† and George Johnson.‡

Wilks, in 1853, thought it might be due to atheromatous change in the vessels; and Traube has conjectured that in renal disease obstruction in the kidneys themselves may be the cause of hypertrophy of the heart.

Doctor Johnson has announced, as the result of his recent investigations, the existence, in the granular form of renal disease, of hypertrophy of the muscular coat of the minute arteries throughout the body. He writes: "The glandular tissues of the kidney suffer in the discharge of their excretory functions, and the organ wastes and contracts. The blood is then rendered secondarily impure by retained urinary excreta, and it is otherwise altered in its composition. This morbidly changed blood excites unusual contraction of the minute arteries, by which the circulation is impeded; the left ventricle, on the other hand, beats with increased force to drive the blood through the resisting arteries. The result of this antagonism of forces is, that the muscular walls of the minute arteries throughout the body, and those of the left ventricle of the heart, become simultaneously and equally hypertrophied.§

This doctrine has been challenged by Sir W. Gull and Dr. Sutton, in a recent communication to the Medico-Chirurgical Society of London,|| and mainly on the evidence afforded by

\* As stated by Quain, *loco antea citat.*

† *Reports of Medical Cases*, 1827, vol. ii., part i., and *Guy's Hospital Reports*, vol. i., 1836.

‡ *Diseases of the Kidneys*, 1852, and *Medico-Chirurgical Transactions*, *passim*, during the preceding six years.

§ *Medico-Chirurgical Transactions*, vol. xxxiii., and *British Medical Journal*, March 7th, 1868, and June 8th, 1872.

|| *The Pathology and Symptoms of the Contracted Kidney, commonly called Gouty or Cirrhused Kidney*, June, 1872.



microscopical examination of the arterial coats. They hold that the change in the minute arteries consists in thickening of the *intima* and *adventitia*, from a deposit of "hyaline-fibroid" material in the arterioles, and "hyaline-granular" in the capillaries; which, in the intertubular and intervascular connective tissue, assumes a fibroid character. This change induces a condition which they propose to designate "arterio-capillary fibrosis." It takes place in the minute arteries throughout the body, but especially in those of the pia mater, and it may be unaccompanied by any change in the kidneys. Hence, according to them, hypertrophy of the heart; which, being due to increased muscular action of the organ, consists in increased development of its muscular structure exclusively, and differs from hyperplasia or connective tissue hypertrophy, the product of irritation, as from the excessive use of alcohol.

Thus, when the heart is found hypertrophied in connexion with chronic renal disease, the primary cause of enlargement of that organ should be sought, not in the kidneys, but in the minute arteries and capillaries, the "fibrosis" of which would constitute the *point de départ* of the series of organic changes, including that of the heart. I am not aware, however, that any examples of cardiac hypertrophy have been adduced, which were traceable to such change in the arteries, to the exclusion of all other possible causes.\*

These pathologists, in short, hold that fibroid change in the kidneys alone is not competent to produce hypertrophy of the heart; that, where this structural change of the kidneys is primary, as in young persons, it may be fatal by uræmia, without having produced either thickening of the arterioles, or hypertrophy of the heart.† The conclusions at which they have arrived are summarized by Sir W. Gull, as follows:‡ "That there is a diseased state coming on about the middle period of life, which is characterized by a morbid formation of fibroid, or

\* *Vide* Dr. Murchison's case, *Transactions of Pathological Society of London*, vol. xxii.

† See an example of this kind, exhibited by Dr. Murchison, and already referred to.

‡ *British Medical Journal*, December 28th, 1872.

hyaline-fibroid tissue, in the arterioles and capillaries: that this is accompanied with atrophy of the adjacent tissues in varying degrees: that this morbid change commonly begins in the kidneys, but may begin primarily in other organs: that the contraction and atrophy of the kidneys is not a cause of the disease, but only part and parcel of the general morbid state: that hypertrophy of the heart is due to changes in the arterioles and capillaries, whereby their elasticity is diminished, and so the blood retarded: it is not due to a morbid condition of the blood itself: that the blood may be fatally affected by disease of the kidneys, without producing any change in the heart, provided the morbid condition of the vessels alluded to is absent: that the causes which lead to these vascular changes are not yet fully elucidated: that they have an alliance with senile conditions, though, probably, they are in their nature distinct."

This much, at least, is certain, that in the class of persons, and at the period of life when cirrhosis of the kidneys is most prevalent, namely, in the gouty, and after the age of forty years, disease of the arteries of the first, second, and third order, consisting in tortuosity of the vessel and rigidity of its coats, is likewise most common; and that with these two conditions, hypertrophy of the left ventricle of the heart, with or without valvular disease, is invariably associated.

It is no less certain that in some cases, no doubt few in number, granular degeneration of the kidneys, and hypertrophy of the left ventricle, may coexist without arterial or valvular disease. This is likewise the opinion of Dr. Grainger Stewart;\* and, further, these changes may occur at a period of life, and under such other circumstances as preclude the supposition that general arterio-capillary fibrosis was present. Case 27 (Marianne Carroll) is a good example of this kind. This girl was only nineteen years of age, and her illness dated back not more than twelve months. The kidneys were granular, weighing two and a-half, and three ounces respectively. The heart weighed nineteen ounces, the left ventricle was greatly hypertrophied; and there was no arterial or valvular disease save recent congestion of the mitral.

\* *British Medical Journal*, November 15th, 1873.

Doctor George Johnson has, after a careful personal inspection, criticized the microscopical preparations offered in support of this theory, declaring that the alleged changes were due, not to a morbid process, but to soaking in glycerine and acetic acid. I do not think his objections have been satisfactorily answered, or that his doctrine has been in any measure disturbed by these investigations.

The condition of muscular hypertrophy was first made out in the minute arteries of the brain, which, owing to the facility presented by them for examination, from the laxity of their areolar connexions, were specially selected for this purpose; but it exists in no less degree in the corresponding vessels of other parts of the body.

Thickening of the muscular element of the walls of the systemic arterioles, as described by Dr. Johnson, is, no doubt, a contributory, although only a secondary, cause of left ventricular hypertrophy. It is but the result of a reaction, or effort at self-adjustment of these vessels, which, subjected for some time to increased pressure from the left ventricle, undergo proportionate development, and acquire a corresponding power of resistance. This resistance, in turn, reacts upon the left ventricle, and determines still further hypertrophy of its walls.

Action and reaction, with alternate increase of muscularity of the ventricle and of the minute arteries, might thus go on indefinitely, were it not for failure of nutrition, and consequent tissue deterioration, in both. In the ventricle this assumes the character of fatty degeneration of the muscular fibre, and induces debility and dilatation; whilst in the arteries it consists in a similar change of the contractile fibre-cells, and atheroma of the middle coat, with consequent liability to aneurismal dilatation and rupture.

Bright found the heart enlarged, without pericarditis or valve-disease, in twenty-nine out of one hundred cases of albuminuria. In seventeen of these cases the left ventricle was the portion of the heart affected; and in the remaining twelve the part is not mentioned.\* Whilst regarding the increased development of the left ventricle in albuminuria as the result of its abnormal func-

\* *Guy's Hospital Reports*, vol. i., 1836.

tional activity, and the latter as due to the stimulating quality of the blood by retained excreta, he considered that this operated, not alone indirectly through the spasmodic resistance to the circulation which it induced in the small arteries, but also directly upon the heart itself.

Jaccoud\* is disposed to accept, in part, the mechanical explanation of hypertrophy of the left heart in renal disease propounded by Traube; but, in many cases, he thinks with Bright, it may be due to the altered state of the blood.

Doctor Grainger Stewart gives the following as the proportions in which hypertrophy of the heart was present in the different forms of Bright's disease.† Of the "inflammatory form," it was present, and apparently referable to the disease of the kidneys exclusively, in the proportion of 40 per cent., the proportion increasing with the progress of the disease up to the last stage, when it amounted to 100 per cent. Of the cases fatal in the first stage, hypertrophy was present in the proportion of 12 per cent.; of those fatal in the second stage, in the proportion of 38 per cent.; and of those fatal in the third stage, or that of contraction, it existed in the proportion of 100 per cent.‡

In the "amyloid form," it existed only in the proportion of 4 per cent. of the cases; and in all these the disease of the kidneys was in its third, or atrophic stage.

Of cases of the "cirrhotic form," 46 per cent. exhibited enlargement of the heart, assignable to no other cause.

Quain states that the left ventricle was hypertrophied in twenty-one cases out of twenty-six of granular disease of the kidneys.§

The connexion between chronic renal disease and left ventricular hypertrophy, as cause and effect, may, therefore, be regarded as well established; and the progress of the latter affection, its consequences, and the collateral changes in other organs and tissues, fully ascertained.

\* *Clinical Lectures on Medicine*, delivered at the Charité Hospital, Paris, 1867.

† *Bright's Diseases*, 1868, p. 41.

‡ *Opus citat.*, second edition, p. 90.

§ *Loco citat.*



Amongst the mechanical causes of hypertrophy of the heart, Roger mentions the pressure made upon it by a deformed thorax.\* This is, no doubt, a not infrequent cause of hypertrophy with dilatation of the right side of the heart, as previously stated by Rokitsansky, owing to the mechanical impediment usually presented to the circulation through the lungs; but the left side is not affected, except secondarily and remotely, and then by dilatation only. \*

Doctor Law maintains that, in the absence of patency of the foramen ovale and of an imperfect septum ventriculorum, hypertrophy of the right ventricle is scarcely ever met with. In the exceptional cases, hypertrophy of the right ventricle is, in his opinion, due to the stimulating action upon it of the arterial blood which finds entrance from the left side.† I have already expressed the opinion that hypertrophy of the right ventricle is very rare. The pathological associations in which it may occur are, no doubt, chiefly those mentioned by Drs. Law and Yeo (*vide* p. 492); but others should be admitted; for example, the pressure of a tumor, aneurismal or cancerous, upon the trunk of the pulmonary artery.

There may, indeed, be hypertrophy of the right ventricle from emphysema of the lungs exclusively, as mentioned by Stokes,‡ and without its being indicated by precordial dulness, or by such only in a slight degree. I have, myself, exhibited before the Pathological Society a heart illustrative of the truth of this remark.

Doctor Clifford Allbutt§ would constitute mechanical diseases of the heart, I think unnecessarily, a distinct class. He states the form which they assume, and their relative frequency of occurrence to be the following:

1. Dilatation of right ventricle.
2. Dilatation of left ventricle.
3. Hypertrophy of left ventricle, or of both ventricles.
4. Chronic inflammation of aorta, and aortic valves.
5. Dilatation of aorta.

\* *Journal of Practical Medicine and Surgery*, November, 1866.

† *Pathological Society*, April 14th, 1866.

‡ *Dublin Journal*, vol. ix., old series.

§ *St. George's Hospital Reports*, vol. v., 1870.

6. Incompetency of aortic valves.
7. Further compensatory hypertrophy of left ventricle.
8. Loss of compensatory power of left ventricle, with rapid failure, and often with mitral regurgitation.

These views are in some respects original; but I cannot coincide in the opinion of the author, that there is anything peculiar in the pathology of these cases, either as to the nature of the morbid changes, or the mode and order of their appearance.

Aneurism of the aorta has been, by many eminent authorities, regarded as a cause of hypertrophy of the left ventricle. Bertin has so regarded it;\* so also have Rokitansky,† and Fuller;‡ and Lebert, Niemeyer, Oppolzer, and Bamberger, on the authority of Professor Axel Key of Stockholm.§ The last named eminent writer, however, declares that aortic aneurism has no such effect upon the heart, however rational it may seem to expect that it should. He has, on the contrary, met with examples of atrophy of the heart in connexion with aneurism of the arch of the aorta. When the valves are healthy, aneurism, in his judgment, exercises no influence upon the heart in regard to its size, form, or condition in other respects, and when hypertrophy or dilatation exists, it is due to an accompanying valvular lesion. The absence of hypertrophy he attempts to explain, by assuming an interrupted pulmonary circulation through pressure of the aneurism upon the pulmonary artery, its branches, or upon the lungs themselves. The tumor, moreover, by abstracting largely from the quantity of blood in actual circulation, tends to diminish rather than to increase the volume of the heart; and lastly, it favours this result, in his judgment, by impairing general nutrition, and inducing a state of anæmia.

But pressure of an aortic aneurism upon the pulmonary artery, or upon the lungs, of such a kind as to obstruct the circulation in them, is certainly an exceptional occurrence. Whilst, as long since shown by Dr. Stokes, aortic aneurism does *not* entail failure of nutrition, or anæmia, except where

\* *Traité des Maladies du Cœur*, p. 350.

† *Pathological Anatomy*, vol. i., p. 163.

‡ *Diseases of the Chest*, p. 659.

§ *Medical Times and Gazette*, June 4th, 1870.

it presses upon and obstructs the thoracic duct. Putting out of consideration, however, the soundness of his reasoning, the statistics furnished by Professor Axel Key are of great value as a contribution towards the settlement of this question. He has had, in all, seventeen cases of aneurism of the aorta; and all, with one exception, situated near the heart. Yet, in no single instance were the walls of the heart hypertrophied; in comparatively few were they even of normal thickness; and in all the remaining cases they were actually thinned, either without any, or with a very partial and localized dilatation of the left ventricle. Instead of hypertrophy, he avers that, taking into consideration the actual volume of the left ventricle, he has found atrophy of the walls of that chamber to exist in the majority of cases, even of large aneurism of the arch of the aorta. Long previously, however, as Axel Key has admitted, Dr. Stokes had declared that the connexion\* of hypertrophy of the heart with aneurism of the aorta, was only accidental. He says: "In some instances, hypertrophy has preceded the aneurism, or there has been a deficient state of the aortic valves; but where the heart or its valves have not been previously engaged, there is no reason to believe that the existence of aneurism, in any portion of the aorta, throws additional labour on the heart, and hence we commonly find a healthy heart coexisting with a vast aneurism."† My own statistics are drawn from a total of twelve cases of aneurism of the intra-thoracic aorta, ten of which engaged the arch, and two the inferior portion of the vessel. Ten of the patients were males, and two females; and in two only out of the twelve, valvular disease existed, and this to a very trivial extent. Seven of the cases were fatal. The average duration of aneurism in these seven, as calculated from the most reliable evidence that could be obtained, having been eleven months and eleven days.

Of these seven, only two, aged thirty-three and fifty-six respectively, exhibited hypertrophy of the heart; and this but in a very slight degree, and affecting *equally* the right and left ventricle. Both these last mentioned cases presented extensive

\* *Ibid.*, June 11th and 18th, 1870.

† *Diseases of the Heart and Aorta*, 1854, p. 579.

and advanced atheroma of the aorta ; the subjects of both were intemperate, and one of them was, moreover, a man of very laborious habits, a porter in the egg-market, and accustomed to lift and carry heavy hampers some distance upon his shoulders. The other, an architect by profession, was likewise of active habits, accustomed to long walks, and to the ascent of high scaffolding in his professional avocation. The aneurism in both engaged the arch of the aorta. In two other cases out of the seven, the heart was in a state of atrophy. The ages of these patients were thirty-four and twenty-eight respectively ; both were intemperate, and in both the seat of aneurism was the descending portion of the aorta, below the arch. The aneurism in the latter of these (William Fay) was of enormous size, displacing the heart and liver, and descending into the abdomen. There was "slight" atheroma of the arch in one of these two, and in the other the vessel was apparently sound, except in the seat of the aneurism. In the three remaining cases of those which were fatal, the heart was normal as to size and capacity. The ages of the patients were, thirty-one, forty, and fifty-four years respectively. Two were temperate (one a female) ; the aorta was "highly" atheromatous in one ; and no note as to the state of this vessel exists in connexion with the other two cases.

From the foregoing statistics I conclude that aneurism of the aorta, in any portion of its course, is alone not capable of giving rise to hypertrophy of the left ventricle ; that it is quite as often associated with atrophy, as with hypertrophy of that chamber ; that it is more frequently associated with a normal condition of the left ventricle, as to capacity and thickness of walls, than with either hypertrophy or atrophy of it ; and, finally, when hypertrophy of either ventricle is actually found in association with aneurism of the aorta, it is, in the case of the left ventricle, either anterior in date to the aneurism, or, if cotemporaneous with it, the result of a different and coexisting lesion, generally valvular. The occurrence of hypertrophy of the right ventricle, in connexion with aneurism of the aorta, is nothing more than a coincidence, the result of obstruction in the pulmonary circulation ; it is in no degree related, as Fuller surmises, to reflected obstruction in the aorta.



The singular combination of symptoms constituting Graves' or Basedow's disease, namely, persistent palpitation of the heart, enlargement and pulsation of the thyroid body, and projection of the eyes, has been by some authors regarded as a cause of hypertrophy of the heart. According to Stokes, the affection consists essentially in functional disturbance of the heart, which may be followed by organic change; namely, hypertrophy and dilatation of the left ventricle. He adduces two examples from the writings of Sir H. Marsh and Professor R. W. Smith of Dublin, in both of which death was the result of intercurrent disease; and in both there was hypertrophy and dilatation of the left ventricle; but both likewise presented valvular disease. In Marsh's case the auriculo-ventricular valves on the two sides of the heart were thickened on the margins, and the auricles were much dilated, showing that reflux upon them had been for some time in operation. In Professor Smith's case, hypertrophy and dilatation existed in a still higher degree, although the valve affected (the semilunar of the aorta) presented only the "slightest appearance" of structural change.\* With coexistent valvular lesion, it must be conceded that the evidence afforded by these two cases is not conclusive in support of the alleged dependence of left ventricular hypertrophy upon the functional excitement of the heart in Graves' disease.

Trousseau regards the affection as "a neurosis which principally affects the heart and the supra-diaphragmatic arteries." He says, the heart may present temporary alterations, similar to those which are met with in pregnancy; and in rare cases the cardiac lesion is permanent when the neurosis has been of long duration. The temporary lesion of the heart in pregnancy, alluded to by Trousseau, is undoubtedly hypertrophy; hence, it would seem that in the rare instances adverted to by him, the permanent lesion was of that character. He has met with hypertrophy in Graves' disease unaccompanied by valve lesion; but, as he judiciously remarks, this condition may have preceded the advent of the disease in question.†

Graves himself considered the cardiac lesion, in the affection

\* *The Diseases of the Heart and the Aorta*, p. 297.

† *Clinical Medicine*, Sydenham Society's edition, vol. i., p. 571-88.

which now bears his name, to be passive dilatation, temporary or permanent.

Niemeyer denies the occurrence of secondary dilatation with hypertrophy of the heart in this affection; but admits that when it is fatal, which is very rarely the case, weakness and gradual dilatation of the heart, followed by anasarca and œdema of the lungs, are the immediate causes of death.\*

Professor Austin Flint has had under observation six examples of this affection, and in one only was there valve-disease. In the remaining five, when first examined, there was no enlargement of the heart; but in one of these enlargement was subsequently produced by long continued functional derangement. He regards the heart affection as the primary lesion, and enlargement of that organ as a usual consequence of the disease when chronic. He gives the case of a female, aged twenty-eight, in which, at first, no evidence of hypertrophy could be detected: after an interval of eight years, however, notable enlargement of the heart had been produced, as judged by displacement of the organ outwards, and increase of precordial dulness. She died a few months subsequently, and the heart was found to weigh twenty ounces; *but the kidneys were fibrous, and weighed fourteen ounces and a-half.*† The words given in italics render this case valueless as evidence of a pathogenetic connexion between Graves' disease and hypertrophy of the heart.

In a future chapter I shall have occasion to discuss at length the pathology of this disease. Here it will suffice to observe, that I regard it as primarily and essentially a disease of debility, and belonging to the class of cardiac neuroses. Organic lesion of the heart, as due to this disease, is exceptional, and when present, and dependent upon it exclusively, I believe it consists invariably in dilatation with thinning of the walls of the heart. I have treated, since I have been an hospital physician, seven cases of the affection, and of all these I have made careful and repeated examination, and kept copious notes. There was no

\* *Text-Book of Practical Medicine*, translated by Humphreys and Hackley, vol. i., p. 374.

† *The Diseases of the Heart*, third edition, 1870, p. 311-17.

death amongst them whilst under my observation, and therefore I have no *post mortem* evidence to adduce. But from physical exploration I am able to state, with certainty, that enlargement of the heart existed only in two out of the seven cases, and in one of these two there was a systolic murmur at the apex, leading to a well-grounded suspicion that structural disease of the mitral valve existed, and was the cause of hypertrophy. In the second of these cases, the disease had lasted eight years, and the physical evidence led to the diagnosis of dilatation, rather than hypertrophy of the heart.

Males are more frequently affected with hypertrophy of the heart than females, in the proportion of two to one, according to Walshe,\* and it is more common in advanced age than in youth, according to the same authority. Vanderbyl found, out of forty examples of hypertrophy, thirty-four in males, and only six in females. The proportion of two to one, in favour of males, may be accepted as an average; a difference which, no doubt, arises from the more laborious duties devolving upon the male sex in all civilized communities; not from any inherent proclivity in sex, as such. As regards individuals, duties which impose more physical labour, and more exposure to extremes and rapid vicissitudes of temperature, entail proportionately greater liability to hypertrophy of the heart.

The rate of progress of hypertrophy is liable to great variety, according to its cause, the habits and age of the patient, and the comforts with which he is surrounded. Hope mentions a case which ended fatally within a year; and another with adherent pericardium and dilatation, in which death occurred in nine months from the date of first reported illness.†

Bertin mentions a case of simple hypertrophy, which he believed to have been developed in fifteen days. (!)

The renal form is that which soonest ends in death, because, when it occurs, formidable disease of another and a vital organ has already attained an advanced stage of progress.

Next in order are those acute forms of hypertrophy from rupture of the aortic or mitral valve. But in none of these cases

\* *Opus citat.*, p. 281.

† *Opus citat.*, p. 278.

is hypertrophy the sole, or even the chief cause of a fatal issue, although it may precipitate that event through pulmonary hæmorrhage, or by rupture of a diseased cerebral artery. It may also promote an unfavourable issue otherwise, by constituting a necessary step towards tissue-degeneration and dilatation of the heart, and failure of the circulation.

If the habits of the patient be dissipated, and thereby calculated to impair nutrition; if, at the same time, they be laborious, to the extent of overtaxing an impaired organ; if the patient be of advanced age, and thereby more prone to degenerative change of tissue; and, finally, if the wants of the body, as to food, raiment, and external heat, be inadequately supplied, the progress of hypertrophy of the heart to a fatal issue will attain its maximum rapidity.

Doctor Gerald F. Yeo exhibited before the Pathological Society of Dublin,\* a morbid specimen, illustrative of the dependence of hypertrophy of the right ventricle upon atheromatous degeneration of the pulmonary artery. A woman, aged thirty-two years, subject to congestion of the lungs, and who had been under observation for six years preceding, was admitted into the Whitworth Hospital for valvular disease of the heart, following acute rheumatism. The lungs exhibited several distinct foci of sanguineous infarction, and the branches of the pulmonary arteries leading to these were plugged with hard laminated fibrin. The pulmonary arteries, to their finest divisions, were dilated and inelastic, and their inner coat was marked with dull yellow patches of atheromatous degeneration. The auricles were dilated, and their appendices were filled with dry crumbling fibrin. The mitral orifice was contracted in an extreme degree, and the mitral valve greatly indurated. The aortic valve was thickened and insufficient. The left ventricle was hypertrophied, and the right ventricle dilated and greatly hypertrophied; its walls were firm, and in some places three-fourths of an inch thick. The aorta was healthy. The tricuspid valve was thickened. Dr. Yeo remarked that engorgement of the lungs, though it may have aided, was of itself incompetent to produce this extreme hypertrophy of the right

\* Meeting, December 14th, 1872.



ventricle, which, in his judgment, was caused by the morbid alteration in the pulmonary artery.

The *symptoms* and *signs* of hypertrophy must be considered separately, in reference to each of the different forms of the affection.

For the differential diagnosis, Corvisart relied mainly on the general condition of the patient, and the character of the pulse. In "active aneurism" (dilated hypertrophy), the patient is strong, and the pulse strong, hard, and vibrating, and there is visible pulsation of the carotids; whilst in the "passive" form (dilatation) there is general debility, and the pulse is feeble, soft, and weak.\*

Morgagni also relied upon these symptoms as evidence of the dilated form; but Bertin held that they belonged not to this, but to the "simple" form of hypertrophy, of which he gives three examples (observations 34, 75, and 76) affecting the left ventricle, and remarks on the absence of lividity of face, capillary engorgement, œdema, and oppression; all of which, he adds, are present in hypertrophy with dilatation, and are due to the latter condition. Hypertrophy of the left ventricle tends to produce cerebral congestion and apoplexy, epistaxis, and congestive inflammation of the eyes; and hypertrophy of the right ventricle tends similarly to the production of pulmonary apoplexy. These forms of congestion he distinguishes as "active;" and apoplexy supervening on them he designates "*coup de sang*."†

In "simple" hypertrophy Bertin says the sounds of the heart are more dull, remote, and prolonged than natural, but limited to the precordium; and in the "concentric" form the sounds are still more masked, and contrast with the force of the impulse.

In excentric hypertrophy, the sounds are louder, clearer, and more extensively diffused than in health, representing the *coup de marteau*, and are audible posteriorly.

Hypertrophy without complication, he declares, involves no alteration of rhythm, but it predisposes to palpitation on exertion; and when intermittence or great irregularity of the heart's action is observed in connexion with hypertrophy, it is due to other lesions with which the hypertrophy is associated, *e.g.*, narrowing of the orifices, or aneurism of the aorta.

\* *Opus citat.*, p. 130.

† *Opus citat.*, p. 283-9, and 350-1.

Respiration, according to Bertin, is not affected by uncomplicated hypertrophy, except when the latter is excessive, and presents a mechanical impediment to the expansion of the lungs; and in the concentric form, when the same embarrassment to respiration should exist as in the case of a contracted orifice. He dwells upon the fact, not previously recognized, that hypertrophy does not, of itself, impede the circulation, save in the concentric form; and that the effects hitherto attributed to it were, in reality, due to a preexisting organic lesion, which gave rise equally to the hypertrophy and to the symptoms ascribed to it.

Hypertrophy of the left ventricle is characterized by pulsation, strongest in the region of the left fifth and sixth costal cartilages; and hypertrophy of the right ventricle by pulsation of greatest force at the lower end of the sternum, and stronger to the right than to the left of that point. The latter is likewise distinguished by "active" hæmoptysis and jugular pulsation or "fluctuation," which, though regarded by Lancisi as indicative of right ventricular hypertrophy, was justly regarded by Bertin as symptomatic rather of dilatation of the ventricle and inadequacy of the tricuspid valve.

The combination of hypertrophy of both ventricles in different forms may be recognized by adverting to the place of greatest intensity, and to the character of the pulsations and sounds of the heart. Hypertrophy of the auricles is characterized, he says, by the muffled quality of the second sound.\*

The authority of Bertin is so justly high, and his opinions so fully representative of the knowledge of his time in this department of clinical medicine, that I have deemed it desirable to state them thus at length, in order to shew in what modern teaching would correct and supplement his rules of diagnosis.

Intermittence and irregularity of the action of the heart, occurring in connexion with hypertrophy, should not be regarded, as justly stated by Bertin, as arising from hypertrophy as such. He was, however, wrong in regarding these symptoms as indicative of narrowing of any of the orifices of the heart, or of aneurism of the aorta. They indicate rather degeneration and dysæsthesia of the heart, and dilatation of the left ventricle. In enumerat-

\* *Traité des Maladies du Cœur*, 1824, p. 350, et suivantes.

ing the topical signs of hypertrophy, Bertin omits all mention of the most valuable of them, namely, displacement of the point of apex-pulsation to the left.

The value of hæmoptysis, as a symptom of hypertrophy of the right ventricle, is certainly very equivocal; many other diseases of the heart are more likely to give rise to it, and notably constriction of the mitral opening. But, even were this not so, it would be impossible to distinguish active from passive hæmoptysis with sufficient precision, for the purpose of differential diagnosis.

The error of attributing the second sound of the heart to the contraction of the auricles, which was common to Bertin, Laennec, and Bouillaud, and led these eminent men and their disciples into many and grave mistakes in physical diagnosis, has been discussed in a previous chapter, and needs only to be mentioned here.

Bouillaud mentions as primary and essential symptoms of hypertrophy, augmentation of the force and extent of the pulsations, and increased loudness of *both* sounds of the heart; and extension of dulness in, and sometimes arching or prominence of the precordium.\*

But exaggeration of the sounds is characteristic of the mixed form only where dilatation is in excess of hypertrophy, as he elsewhere admits. In the simple form, the sounds are dulled. Percussion-dulness is not necessarily increased, for example, where the anterior edges of the lungs are emphysematous, and overlies the pericardium; and prominence of the pericardium is not only possible in acute pericarditis with copious liquid effusion in the child, but infinitely more common than it is in connexion with hypertrophy at any period of life. He adopts, from his teacher Bertin, the error of attributing derangement of rhythm to stenosis of one or other of the orifices of the heart, and omits, as Bertin did, to mention the actual cause of irregularity, namely, degeneration of the muscular substance of the heart. Laennec states that, whilst moderate hypertrophy of the ventricles causes dulness of the first sound, and exaggerates the natural rhythm of the heart by prolongation of the long pause,

\* *Traité Clinique*, tom. ii., p. 440.

extreme hypertrophy prolongs the systole, and either completely masks the first sound, or admits only a faint murmur like that of respiration. There is an obscure and distant movement which gradually increases, lifts the ear, and gives the sensation of a shock. The ventricular contractions run almost into one another, and there is scarcely any interval of repose, the second sound being curt, and almost or entirely lost.\*

As symptoms of hypertrophy of the left ventricle, Laennec mentions a full and strong pulse; strong and visible impulse; diminution or absence of percussion-resonance in the precordium, and florid complexion; but adds, with truth, that none of the symptoms are constant. Laennec, like Bertin, omits all mention of displacement of the apex and extension of percussion-dulness to the left, as signs of hypertrophy of the left ventricle.

According to Hope, prominence of the precordium and "a double jogging impulse" are symptomatic of an hypertrophied heart, fixed by adhesion of the pericardium.† He truly remarks that the pulse in hypertrophy, complicated with "valvular and other lesions," is liable to many and great modifications, which represent rather the complications than the hypertrophy; but in left ventricular hypertrophy of the simple form, and free from complication, it is regular, strong, full, and tense; it swells gradually and with force, expands largely, and dwells under the finger, and in anæmic subjects it is accompanied with a thrill. In hypertrophy with dilatation, provided the former condition predominate, these characters of the pulse are still more decided; but when dilatation is in excess of hypertrophy the pulse is soft and compressible. In concentric hypertrophy the pulse is tense, but small; and where this condition exists in an extreme degree, it is weak and small.

He holds that dyspnœa, venous congestion of the lips, and passive hæmorrhage, may be caused by uncomplicated hypertrophy, through embarrassment of the capillary circulation; and that angina, although characteristic of no particular lesion of the heart, is often associated with hypertrophy.

The impulse of the heart is strong, heaving, and sustained, in

\* *Traité d'Auscultation Médiate*, 1826, tom. ii., p. 411.

† *A Treatise on the Diseases of the Heart*, etc., p. 238.



proportion to the degree of hypertrophy and accompanying dilatation; and in both the excentric and simple forms Hope notices, with especial emphasis, the existence of a "diastolic impulse," for the discovery and interpretation of which he justly claims credit.

In reference to this sign, he says: "It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back terminates in a jog, or shock, occasioned by the refilling of the ventricles, and constituting the diastolic impulse in question. It is stronger, *cæteris paribus*, in proportion as the heart is thicker and more capacious. Accordingly, I have found it strongest in hypertrophy with dilatation; but it may also be very considerable in simple hypertrophy. In the healthy heart it is not perceptible; neither is it in dilatation without hypertrophy."\*

Hope agrees with Laennec in thinking the ear, applied to the stethoscope, the most delicate medium for the appreciation of cardiac impulse. Jugular pulsation he regards as a sign of dilated hypertrophy of the right ventricle, maintaining that the double venous fluctuation is due, the lesser, to the wave of blood urged on by the contraction of the auricle towards the distended ventricle, and thence reflected upon the superior cava and its tributaries; and the greater, to the sudden and powerful contraction of the ventricle upon the mass of blood filling its cavity, and the consequent repulsion upon the auricle, and through it upon the jugular veins, of a column of blood, equivalent in sectional area to the surface of the tricuspid valve thus suddenly and forcibly raised by the contraction of the ventricle.

The heart, he says, occupies a low position in hypertrophy, except when fixed by adhesion of the pericardium, and the sounds are modified according to the form of hypertrophy actually present. Thus, in simple hypertrophy the first sound is dull and prolonged in proportion to the degree of thickening of the walls, whilst the second sound is feeble, and the interval of repose is shortened because of the increased length of the first sound.

In concentric hypertrophy, he says, both sounds are proportionately weaker; and in the simple and concentric forms alike,

\* *Opus citat.*, p. 271-2.

the first sound is scarcely audible at the upper part of the sternum and beneath the clavicles, whilst the second is distinctly heard. Where both ventricles are thus altered, the transmission of the sounds is restricted to the narrowest limits.

In hypertrophy with dilatation the sounds are loudest, and during palpitation they attain their utmost intensity. The first is abrupt at its commencement, and prolonged, whilst the second is simply intensified; both are extensively transmitted through the chest, and distinctly audible at all points of its surface, even behind, especially in children and thin persons.

Where dilatation predominates, the first sound, he says, is not so loud or prolonged as in the preceding variety, whilst the second is louder than natural, but otherwise unaltered.

Both the impulse and the sounds are diminished by general debility, induced by exhaustion from any cause; by acute and chronic affections of the lungs; and by approaching dissolution.

I have already expressed the opinion, that precordial prominence is not characteristic of hypertrophy, because proportionately more frequent in hydro-pericardium; and that elevation of the point of apex-beat, without any discoverable cause of excentric pressure on the heart, is indicative of adhesion of the pericardium exclusively. The "double jogging impulse" in no respect differs from the double impulse, systolic and diastolic, to be presently noticed.

Symptoms of venous engorgement are certainly not characteristic of uncomplicated hypertrophy in any of its varieties, quite the reverse; and dyspnœa is so only in a qualified sense; namely, when the hypertrophy is excessive, and incommodes the lungs by its mere bulk and pressure; or when it is induced by unwonted physical effort of any kind. The capillaries are not engorged as a consequence of uncomplicated hypertrophy. When they are in that condition in connexion with hypertrophy, the cause of the hypertrophy is also the cause of the capillary engorgement; *e. g.*, valvular disease. In the early stages of hypertrophy the capillaries are not engorged, but in a state of active hyperæmia. When, however, in the latter stages, the heart begins to fail, passive congestion of the capillaries and veins ensues: a change due, not to hypertrophy, but to the atrophic and degenerative

changes of tissue which constitute its inevitable sequelæ. Then, for the first time, the pulse is noticed to be irregular, and dropsical effusion appears. I regard as of no weight against this view the case of alleged concentric hypertrophy, causing dropsy and other consequences of capillary and venous congestion, adduced by Hope,\* in the absence of positive evidence that the kidneys were not diseased.

The interpretation of the "diastolic impulse" given by Hope is, in my judgment, only partially correct. Such impulse is present only where hypertrophy with dilatation exists; never in simple hypertrophy. His explanation of the phenomenon implies two coincident and essential conditions: a "sinking back" of the heart, and diastole of the ventricles. I believe the latter constitutes the sole cause; the former having no existence, and, therefore, no share in the production of diastolic impulse.

It is no less perceptible when the patient lies face downwards, and no "sinking back" can take place. Further, Hope's theory implies that diastolic impulse takes place at the *end* of ventricular diastole, and coincides, therefore, with auricular systole. But, however plausible this doctrine may seem, as introducing the element of active impulsion of blood from the auricle, it is opposed to observation, which shows that the diastolic impulse coincides with the second sound of the heart, and therefore with the *first* period of ventricular diastole. My view is, that the hypertrophied, dilated, and dense walled ventricle, returning by recoil to a state of complete diastole, and becoming at the same moment suddenly and rapidly filled with blood by influx from the auricle, attains thereby dimensions in excess of the antero-posterior diameter of the mediastinum; and partly by its diastolic recoil, and in part by the excentric pressure thus exercised upon its internal surface, it is brought suddenly into violent collision with the precordium, and becomes the cause of sensible impulse. It is, I believe, pathognomonic of dilated hypertrophy of an unsoftened left ventricle. With an exaggerated systolic impulse, this phenomenon constitutes the "double jogging impulse" previously mentioned, as indicative, according to Hope, of hypertrophy, with adherent pericardium; but, as I

\* *London Medical Gazette*, September 5th, 1839.

think, erroneously so interpreted. Neither the explanation offered by Hope of jugular pulsation, nor his interpretation of that phenomenon, is, to my mind, satisfactory. I believe it to be due, as already stated, to actual reflux from the right ventricle; and that it is, therefore, pathognomonic of tricuspid regurgitation. There are noticeable in the external jugular veins, two distinct movements in such cases: one, a single reflux wave of considerable magnitude, coincident with the systole of the ventricles and the carotid pulse, and all but abolished by a full inspiration; and the other, a series of ripples, likewise reflux, but in no degree influenced by respiration, alternating with the larger fluctuations, and occupying the whole interval between them. Both these movements may be promptly arrested by gentle pressure upon the vein, immediately above the clavicle. The former I believe to be due to reflux from the ventricle during its systole; and the latter, to reflux from the auricle during the series of feeble undulatory contractile movements performed by it previous to its final systole.

The systole of the auricle, properly so called, which immediately precedes the systole of the ventricle, is not productive of distinct venous reflux. The tricuspid orifice being thrown open at the moment of auricular systole, the blood passes by the larger opening into the ventricle; it is urged by the onward contraction of the auricle, and its course is favoured by gravitation, more freely than by the smaller aperture and against gravity, back upon the cava and jugular veins.

It is possible, however, that a reflux jugular wave may be actually produced by right auricular systole; but it would be necessarily merged in that of ventricular systole which immediately follows, and therefore not separately recognizable.

It may be and has been urged against this explanation of venous pulse in the neck, that it necessarily implies the concurrent development of tricuspid reflux murmur. But where the distention of the ventricle is such as to render the tricuspid valve incompetent, its power of contraction is manifestly insufficient to educe a murmur. Hence, the extreme rarity of tricuspid regurgitant murmur; so rare, indeed, is it, that I have never heard it except in cases, very few in number, in which incom-



petency was the result of valvular lesion without distention of the ventricle.

In speaking of an abbreviated interval of repose as a collateral sign of hypertrophy, Hope does not make it clear to which of the two intervals he refers. The first interval is shorter; but the second, or that following the second sound and preceding the first, is longer than natural, as stated by Laennec, because the prolongation of the first sound is effected by *suffix*, not by prefix; ventricular contraction not anticipating, but *transgressing*, its normal period.

I have not met with an example of the double systolic impulse which Walshe notices as a sign of excentric hypertrophy.\* I have occasionally observed, in the early stages of retrograde metamorphosis of an hypertrophied heart, an abortive pulsation or tick, not represented in the radial pulse, to immediately succeed the normal impulse at the precordium. But this is symptomatic, not of hypertrophy itself, but of the retrogressive changes consecutive to that condition.

Neither can I, in view of the negative and positive signs, admit with that eminent observer, the possibility of mistaking excentric hypertrophy in a narrow chested subject, for pleural effusion.

In extreme cases of dilated hypertrophy of long duration, and also in early youth, Walshe has noticed bulging of the precordium, divarication of the ribs on the left side from the second to the eighth, and percussion-dulness to the same extent; the head of the observer, the body of the patient, and even the bed being visibly shaken whilst the stethoscopic examination was being conducted. This, I have likewise witnessed, but only in childhood (case T. D.). I cannot agree with him in the opinion, that reduplication of the sounds possesses no special significance; neither can I accept his view of its dependence, in regard to *both* sounds, upon inequality of strength in the two ventricles.† I believe reduplication of the first sound, as already stated (p. 165), positively indicates hypertrophy with dilatation; or derangement of cardiac innervation; and that it depends upon

\* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 32.

† *Opus citat.*, p. 283.

disturbance of synchronism between the systole of the ventricle, and the closure of the corresponding auriculo-ventricular valve.

Reduplication of the second sound is caused by unequal repletion of the two ventricles, and consequent derangement of synchronism in their evacuation, and in the consecutive reaction of the aorta and pulmonary artery; the closure of the semilunar valves being postponed on the side of greatest ventricular distention.

The doctrine that hypertrophy is capable, *per se*, of inducing murmur at the aortic or mitral orifice by the mere force of contraction to which it may give rise, is quite unsupported by the result of my observation. I have repeatedly, however, encountered such a phenomenon; but at the mitral orifice only, in hearts, whether hypertrophied or not, dynamically weakened by softening of the ventricular walls.\* In no single instance have I met with aortic systolic murmur not traceable to some definite organic lesion of an obstructive kind, either at the orifice or in the root of the aorta, which was not manifestly of hæmic origin. I cannot, therefore, believe in the alleged occurrence of aortic systolic murmur from derangement of proportion between a dilated ventricle and its orifice of exit; or from derangement of the line of axis from the orifice of the ventricle to the aorta, till conclusive evidence be adduced of its actual occurrence.

Amongst the general symptoms of hypertrophy, Walshe mentions paroxysms of dyspnœa in case of much dilatation, valvular obstruction, or pulmonary disease. Dyspnœa, occurring in paroxysms in connexion with hypertrophy, I should regard as evidence of dilatation and rigidity of the aorta.

Particular tinting of the face, frequent headache, rushing noises in the ears, drowsiness after meals, startings in sleep, and epis-taxis, although collectively or singly often present in hypertrophy, are quite as often absent; hence they can be regarded only as *presumptive* symptoms of that condition.

Enlargement of the thyroid body is assuredly not a consequence, and therefore not a symptom of hypertrophy. It is commonly associated with excitement of the heart, which may or may not be followed by hypertrophy; but in relation to both it stands as cause, not as effect.

\* See Chapter III., p. 281.

In connective tissue hypertrophy, Quain states that the impulse is strong and heaving, and the first sound dull; and in that due to accumulation of fat, which he admits as a special form, without adequate reason in my judgment, the sounds are sharp.\*

In the latter condition the sounds are, no doubt, in most cases sharp at the base, owing to the usually concomitant change of atheroma of the aorta. But the apex-pulsation is feeble, and the apex-sounds faint, and of low pitch, except in the advanced stage of concomitant degeneration of the muscular fibres, when the sounds, though faint, may be likewise sharp and brief.

The symptoms and the signs† of hypertrophy may be divided into those which are *presumptive* or *equivocal*, and those which are *positive*; and each member of these two groups must be considered separately in relation to the different forms of hypertrophy specified at page 462.

## EQUIVOCAL.

*Symptoms.*

Florid or congested face.  
 Liability to headache in recumbent posture.  
 „ to rushing noise in the head on stooping, or after exertion.  
 „ to violent palpitation after exertion.  
 „ to epistaxis.  
 „ to vertigo.  
 Habitual shortness of breath on exertion.  
 Throbbing of the carotid arteries.

*Signs.*

Extension of precordial dulness.  
 Masking of cardiac sounds.  
 Extensive diffusion of cardiac sounds.

## POSITIVE.

Large, full, and strong pulse.  
 Visible heaving of the precordium.

Displacement of the apex to the left, in the absence of excentric pressure.  
 Extension of area of apex-pulsation.  
 Heaving precordial impulse, perceived by the application of the hand.  
 Extensive diffusion of impulse.  
 Prolonged, dull, and distant firstsound.  
 Reduplication of first sound.  
 Double impulse.  
 Sphygmographic tracing of hypertrophy.

\* Lectures, *loco citat.*

† For convenience sake I use the word "sign," as contradistinguished from symptom, throughout this work, as indicating sensory evidence discoverable by auscultation, percussion, or palpation.

It is unnecessary to discuss here the equivocal symptoms and signs, which have but a casual connexion with hypertrophy; each of which may be absent where hypertrophy exists, or may be present in its absence, and due to other causes.

Of the positive symptoms of hypertrophy, the large, full, strong, and sustained pulse is amongst the most valuable; it is distinguished by the *combination* of these qualities from the pulse of all other morbid states whatever, and is, therefore, pathognomonic of left ventricular hypertrophy. It is liable to great variation as to firmness, according to the state of the walls of the artery examined; but, it is always of high tension, except in the stage of retrogressive change of tissue.

The absence of pulse of this character would not be of equal value as negative evidence in regard to the heart generally; because the right ventricle alone may be hypertrophied, and yet the pulse may present characters the opposite of those just mentioned.

In simple hypertrophy of the left ventricle, the pulse is less full and sustained, but no less strong, than in the dilated form. The difference, therefore, is only of degree, as between the simple and dilated forms of the affection on the left side of the heart.

Hypertrophy of the right ventricle is nearly always of the dilated form, and the radial pulse exhibits characters derived from an obstructed pulmonary circulation, viz., those of smallness, weakness, and occasionally even of intermittence or irregularity.

Visible heaving of the precordium is no less significant of hypertrophy, in the assumed absence of aneurism or other tumor behind the heart. When the left ventricle alone is affected, the movement will be perceived towards or beyond the line of the left nipple; and where the right alone is engaged, it will appear, but less distinctly, in the inferior sternal region, and to a variable distance beyond the right edge of that bone. If both ventricles be in the state of hypertrophy, the movement of impulse will be visible over the entire extent of the precordium, but most distinctly at the point of apex-pulsation; and in the dilated form it will be of an undulatory character.

As a single sign, displacement of the apex to the left of its normal position might be due equally to hypertrophy and dila-



tation of the left ventricle; and the same may be said of enlargement of the area of apex-pulsation. But, associated with heaving impulse at the left precordium perceptible to the hand, these signs, single or united, become pathognomonic of left ventricular hypertrophy. The degree of displacement of the apex to the left, and of extension of its area, will, in the association just mentioned, measure with approximate accuracy the enlargement of the left ventricle; and its downward and leftward displacement, the expansion and elongation of the organ so characteristic of hypertrophy from incompetence of the aortic valve. In determining the extent of apex-pulsation I make use of my index and middle fingers as a pair of callipers, and with sufficiently accurate results.

Heaving precordial impulse is no less distinctive of hypertrophy; it is of the nature of a prolonged swell or upheaving of the chest-wall, by the shock and gradual uplifting of a powerful solid body immediately behind it. In simple hypertrophy the shock is more abrupt and localized, and the lifting of the chest-wall less decided than in the dilated form. In excentric hypertrophy of extreme degree, the shock of cardiac impulse is so powerful as visibly to shake the body of the patient, and the head of the auscultator, as correctly stated by Hope. I have seen it communicated to an enlarged liver, and I have heard gastric tinkle, corresponding to the systole of the ventricles, caused by it. It is, moreover, extensively diffused over the front of the chest, being perceptible to the hand far beyond the normal limits of the precordium.

The first sound of the heart is strong, but dull, prolonged, and distant in all forms of hypertrophy. In simple hypertrophy the dull and distant characters of the sound predominate; and in the dilated form, prolongation and strength, with greater clearness, are the distinguishing qualities. The second sound is less distinctly audible at the apex in both forms than it is in the healthy heart; louder, however, in dilated than in simple hypertrophy. At the base, the second sound is usually distinct and normal; but in many cases of excentric hypertrophy it is morbidly loud, sharp, and clear, for some distance in the line of the ascending aorta. This is, in some degree, due to the force of im-

pulsion, and usually also to antecedent or consecutive atheroma of the coats of that vessel.

Both sounds are extensively transmitted in dilated hypertrophy, especially in young and thin subjects. In such I have heard them at all points of the chest, even posteriorly. A solidified lung would seem to intensify, by transmission, the sounds of the heart in these cases.

Reduplication of the first sound is so intimately associated with simple or excentric hypertrophy, that if carefully sought, it will be found in every such case, anterior to consecutive deterioration of tissue. It consists, as already stated, in resolution of the first sound into its constituent elements of impulse and valve-tension; and is due to the abolition of simultaneousness of these events, and the interposition of an appreciable interval of time between the impulse by which the systole of the ventricles is inaugurated, and the tension of the auriculo-ventricular valves, which occurs at its acme.

The suddenness and energy with which ventricular contraction is initiated in simple hypertrophy, and the ordinary rate at which its acme is attained, explain at once the early occurrence of impulse, and the apparent postponement of valve-sound; whilst, in excentric hypertrophy, the derangement is due to postponement of valve-tension, arising from the protracted process of ventricular evacuation. In both, the exalted contractile force acquired through increased parietal development, accounts for the emphasis with which the two elements of the first sound are pronounced. The absence, in a simple dilated ventricle, of contractile power adequate to the production of audible impulse, may account for the absence in such cases of double first sound. Hence, likewise, double first sound has rarely, if ever, its source in the right ventricle; for, even when hypertrophy is associated with dilatation of this chamber, an event of very rare occurrence,\* dilatation is so much in excess that audible impulse is scarcely developed, and inadequacy of the tricuspid valve is a

\* Bertin has, however, reported a case (88) in which the walls of the right ventricle were from eleven to sixteen lines thick. Bouillaud gives one (76) in which the thickness was eight to ten lines, and a second (77) in which it was twelve lines; and Hope gives an example of it, measuring six to seven lines, in a girl nine years old.

necessary concomitant ; hence, absence of valve-click, and therefore, of double first sound.

I have already (p. 499) stated my opinion as to the cause and diagnostic significance of the "double jogging impulse" of Hope. It will suffice here to repeat, that I regard it as identical with double impulse of systolic and diastolic rhythm, as described by that author ; and as evidence, not of simple hypertrophy with or without adhesion of the pericardium, but of hypertrophy with dilatation of the left ventricle. Dilatation of the right ventricle, as just stated, is rarely associated with hypertrophy of its walls. Hence, likewise, double impulse rarely has its origin in this chamber. I am not personally cognizant of a single example of the kind.

A sphygmographic tracing would, of course, afford conclusive evidence of hypertrophy of the left ventricle.

Hypertrophy of the right ventricle must be judged of by other evidence. The condition usually met with and designated hypertrophy, is that of dilatation of its cavity with normal thickness of its walls.

Veritable hypertrophy with dilatation of this chamber is, however, of occasional occurrence. It has been met with in association with long continued mitral obstruction ; with pulmonary emphysema ; and with primary hypertrophy of the left ventricle from over physical exertion, and subsequently complicated with chronic bronchitis, in well nourished but gouty subjects.

General hypertrophy of the right ventricle may likewise be witnessed in connexion with the exceedingly rare lesion of obstruction at the orifice of the pulmonary artery, without perforation of the ventricular septum or patency of the foramen ovale. Where direct communication between the right and left side of the heart exists, by imperfection of the septa, the right ventricle may, according to Dr. Law, undergo hypertrophy from the circulation of arterial blood through its cavity.

Doctor Sutton holds that in this condition of the right ventricle, systolic *bruit de soufflet* may be developed at the tricuspid orifice by incompetency, without structural disease of the tricuspid valve.\* It is, he says, characterized by being audible at

\* *London Hospital Reports*, vol. iv.

the ensiform cartilage, or a short distance to the left of the sternum at the level of the fifth and sixth costal cartilages, but *not* at the left apex or left scapula. He gives the details of eight cases in which he had personal knowledge of its existence, and in which he examined the body after death. The tricuspid orifice was dilated in all these cases; but, in several of them, likewise, the edges of the valves were rough. In a ninth example given, tricuspid reflux murmur existed, although, as proved by examination of the body, the right ventricle was simply dilated, but the valves were uneven, and manifestly incompetent. I have already stated that, even where veritable tricuspid reflux was proved to exist by jugular venous pulsation, in cases of distention of the right chambers of the heart with or without hypertrophy of the right ventricle, I have never heard tricuspid regurgitant murmur. I have, in a few cases, heard it where the right chambers were not engorged; but the valves had been rendered incompetent by roughening of their edges. I venture to suggest, and the facts as furnished by Dr. Sutton do not absolutely forbid this view, that, in those of his cases in which a rough state of the tricuspid valves existed, regurgitation and murmur would be thereby adequately explained; whilst in the others, the murmur may not have been adynamic and mitral; a murmur which is rarely audible in the left axilla, and never at the left scapula. Addison, as stated by Dr. Sutton, did not believe in tricuspid regurgitant murmur, and Hope and Walshe regard it as very rare.

The signs by which this condition of the right ventricle is manifested are few; and, with one exception, indifferently pronounced. The exception alluded to is the existence of strong impulse at the lower sternum and ensiform cartilage, in the absence of pulmonary emphysema, or other cause of displacement of the heart to this locality. The impulse in question is diffused, and of moderate strength rather than heaving; apex-pulsation is not perceptible, unless the left ventricle be also in a state of hypertrophy, because the apex is thrown backwards by the increased development of the body of the right ventricle.

Pronounced, and amply sufficient for the purpose of diagnosis, as are the signs of hypertrophy discussed in the last pages, the



evidence of its existence is not usually limited to these signs. There are present, in addition, in the majority of cases, the signs of valvular lesion, obstructive or regurgitant, which must lead, and within a brief period, to hypertrophy of the corresponding ventricle. The existence of a well pronounced systolic murmur at the right base of the heart, traceable through the arch of the aorta and into the carotids, with or without diastolic murmur in the same situation, should suggest the coexistence of left ventricular hypertrophy. A single and diastolic murmur here would have a like significance. When aortic diastolic murmur exists, the apex of the heart will be found to have undergone displacement downwards as well as towards the left. Systolic murmur having origin in the aorta, above its orifice, and distinguishable from obstructive lesion at the mouth of the vessel by the higher level of its point of greatest intensity, by its non-transmission into the cervical arteries, and by the coexistence of rigidity and tortuosity of the superficial arteries, will be likewise found associated with the positive signs of hypertrophy of the left ventricle.

Mitral regurgitation also leads inevitably to hypertrophy of the left ventricle. Therefore, when apex systolic murmur exists, the distinctive signs of hypertrophy should be looked for. Not so with mitral obstruction. In connexion with this lesion, if unassociated with mitral inadequacy, or other cause of hypertrophy, the left ventricle is either strictly normal in all respects, or somewhat reduced in capacity, but never thickened in its walls.

The existence, therefore, of simple presystolic murmur at the apex, would be *primâ facie* evidence of the non-existence of hypertrophy of the left ventricle; and the absence of the positive signs of that condition, already mentioned, would confirm the negative diagnosis.

The *pathology* of hypertrophy is scarcely on a level with our knowledge of its diagnosis.

Senac, Morgagni, and Corvisart, laid down the principle that the heart became hypertrophied, like the external muscles, under the stimulus of increased work.

Corvisart distinguishes the induration of "active aneurism" from that arising from thickening of the connective tissue of the

heart,\* and gives an example (Observation 27) of the latter kind, in which the pulse was small, hard, and slightly irregular, whilst the heart's action was strong and tumultuous; thus representing the want of harmony between the pulse and the heart, usually regarded as characteristic of aortic obstruction. At the necropsy, all the chambers of the heart were found dilated, rigid, and resonant, and the aortic orifice contracted.

Quain has recently† given a minute description of this form of hypertrophy, in the recognition of which he was, however, long anticipated by Corvisart, as above shown.

Corvisart also supplies an example of cartilaginous alteration of the heart (Observation 28), in which the symptoms were identical with those in the former case, and the pathological change, as he avers, only more advanced.‡ Bouillaud gives several such.§

A condition of the heart corresponding to the description of fibroid cartilaginous change above given, must be extremely rare at the present time. I have not met with a single example of the kind, and I incline to think that its rarity has some relation to the less heroic treatment of hypertrophy now practised.

Rindfleisch attributes fibroid transformation to diffuse parenchymatous inflammation of the muscular substance of the heart; of which he gives an example in a syphilitic subject.|| The heart was rigid, somewhat violet in colour, and in section iridescent. The cut edges were nearly translucent; the consistence that of caoutchouc, but unyielding, so that the fibres would tear before stretching. There were numerous ecchymoses beneath the endo- and pericardium, whilst the blood-vessels of the heart were empty. The interior of the muscular fibres was occupied by fine granules, which were accumulated in fusiform heaps around the nuclei; the fibres exhibited cross rents, the result of mechanical tearing.

The "fibroid patches" of the same writer are likewise regarded by him as of inflammatory origin, and engaging the substance of the heart by extension from the endocardium. The description

\* *Opus citat.*, p. 146.

† Lumleian Lectures, 1872.

‡ *Opus citat.*

§ *Ibid.*, p. 152.

|| *Manual of Pathological Histology*, Sydenham Society's edition, vol. i., p. 275.

just given corresponds to that of "fibroid transformation" of the heart published by Ormerod many years ago;\* but it does not appear that hypertrophy existed in any of these cases.

Sir W. Jenner has shown† that chronic congestion of the heart may lead to "induration," "toughening," and thickening of its walls by interstitial deposit of lymph, and ultimately to permanent dilatation. The walls so affected, though thin, do not collapse on section. The tissue is either dark, or pale, close grained and leather-like, according to the stage of induration attained.

Under the microscope the striæ are seen to be imperfectly marked in distinct foci; the fibres firmly cohere; and between and within them protein granules are visible. Droplets of olein, and fibres in a state of veritable fatty degeneration, are likewise seen in a few places, and when such centres have multiplied and coalesced, softening of a previously indurated heart is the result.

Congestion of the heart, to lead to these results, must be gradually developed and long continued, but intermittent; and the heart must have been, at the same time, actively functioning. Six cases are given by Sir. W. Jenner, in all of which the patients had suffered from chronic but intermittent congestion of the thoracic and abdominal viscera; and after death the heart, liver, and kidneys were found to have undergone the changes above described. It is noteworthy that in three of these cases the mitral orifice was much narrowed.

In the further progress of fibroid change of the heart its substance may become rigid and resonant. Corvisart recorded an example of this kind, in which the heart, when struck, resounded like horn. Laennec met with a similar example. Burns‡ saw the ventricles converted into bone, like the vault of the skull. And Broussais gives two cases in which the heart was as firm as a cocoa-nut shell.

In reference to the prevalent opinion that in hypertrophy the muscular fibres have become actually thickened, Rindfleisch

\* *British Medical Journal*, 1863.

† *Medico-Chirurgical Transactions*, vol. xliii., 1860.

‡ *The Diseases of the Heart*, p. 131.

observes that he has failed to discover any such change; and he intimates his opinion that hypertrophy is due to excessive cleavage of the fibres, by tension of their lateral branches during contraction.

I am not in a position to offer a positive opinion upon this doctrine. It seems to me doubtful that such a condition of the fibres could exist without lessening the density of the heart; yet, as is well known, the hypertrophied heart, previously to retrogressive change, is actually more dense than the healthy organ.

The alleged *consequences* of hypertrophy of the heart, respecting some of which well grounded doubts may be entertained, are the following:

Dyspnœa.

Dropsy.

Dilatation of the aorta.

Apoplexy, cerebral and pulmonary.

Fatty degeneration.

Gangrene.

That dyspnœa is not a necessary consequence of hypertrophy of the heart, is shown by its absence in pedestrians, pugilists, sledgers, oarsmen, etc., whose habits or occupation require the greatest and most protracted muscular efforts, and the highest breathing capacity. The compatibility of hypertrophy of the heart with great respiratory capacity, is likewise exemplified in the racehorse and greyhound. The heart of the celebrated greyhound, "Master Magrath," weighed 9·57 ounces avoirdupoise;\* and as the animal had won its last prize, the Waterloo Cup, only ten months previous to his death, and only two months before his death he beat, with facility, a kennel-companion in a trial coursing match; and as, in the absence of valvular, renal, and arterial disease, no cause of hypertrophy could be assigned, except the great and long sustained muscular efforts to which the dog had been accustomed, the conclusion seems inevitable, that it had coursed for and won in at least its last great prize, with a heart greatly in excess of the normal dimensions of that organ.†

\* Professor Haughton, in *British Medical Journal*, January 20th, 1872.

† The weight of the dog's body in coursing condition was 54 lbs.; that of the heart after death was 9·57 ounces avoirdupoise. The heart was, therefore, threefold in excess of the normal proportion of heart-weight to body-weight.



In the ordinary course of events, however, a period will certainly arise, if death do not intervene, when, owing to retrogressive or degenerative changes of its muscular tissue, the heart will fail as the circulating centre; and vascular congestion and dyspnœa will inevitably follow. But, in all such cases, hypertrophy is a predisposing, not an immediate, cause of dyspnœa. The occasional paroxysms of cardiac dyspnœa which Corvisart witnessed in hypertrophy, and which he conjectured to have been due to "temporary fulness of the cerebral vessels, or those of the lungs,"\* were manifestly of different origin. Paroxysmal dyspnœa of this character, as I hope to show in a future chapter, arises from dilatation of the aorta: a condition by no means infrequent in connexion with hypertrophy of the left ventricle, as the result of either concomitant or consecutive disease of the coats of that vessel. It is conceivable that a heart which had attained extraordinary dimensions, might, by its mere bulk, prevent the full expansion of the lungs, and so diminish respiratory capacity.

*Dropsy*, when consecutive to hypertrophy of the heart, is always due to adynamia or congestion from secondary dilatation, or to fatty change. Dropsy of this kind should, however, be carefully discriminated from renal anasarca associated with hypertrophy of the heart consecutive to renal disease; it may be readily distinguished from the latter by the appearance, simultaneously with the dropsy, of venous and capillary engorgement, together with the physical signs of failure of the heart; whereas, in the renal form, the dropsical effusion long precedes venous congestion and irregularity of the heart and radial pulse. Where fatty disease of the heart, primary or secondary, or of the kidneys, is the cause of anasarca, I have noticed that the scrotum and penis are swollen at a very early period of the disease. I cannot, however, venture to explain the connexion.

*Dilatation of the aorta* is a direct result of dilated hypertrophy of the left ventricle, and may certainly occur without being preceded by disease of the coats of that vessel. I have met with many examples confirmatory of this statement. It would seem

\* *The Diseases of the Heart*, Hebb's edition, p. 164.

as if the enlarged and powerful ventricle had operated injuriously upon the aorta, partly by its intrinsic force of contraction, and partly by the large mass of blood, out of proportion to the normal calibre of the vessel, projected into it at each act of systole. In consequence of this repeated surcharge and over distention, the walls of the aorta soon lose their elasticity, and the vessel remains permanently enlarged.\* The usual changes of tissue consecutive to malnutrition are quickly set up in the coats of the aorta, after dilatation has been established. Over-distention of the vessel causes increased density of its walls; and from this follow, obstruction of the *vasa vasorum*, paresis of the trophic nerves, failure of capillary circulation, and arrest of nutrition in the walls of the aorta. Retrograde metamorphosis and atheromatous change are the results. Thus, whilst atheromatous degeneration of the arterial coats is usually primary and general, and constitutes a *cause* of hypertrophy of the left ventricle, in this instance it is secondary and local, and one of the *effects* of hypertrophy. But dilatation of the aorta may, and frequently does, lead to the further result of incompetency of the aortic valve, by simple derangement of proportion between it and the mouth of the vessel. In such case, two new factors of hypertrophy of the left ventricle are introduced; namely, atheroma of the coats of the aorta, and inadequacy of its valves. The progress of hypertrophy is proportionately accelerated, till such time, usually not distant, as retrogressive softening of its walls shall have been set up.

Dilatation, by yielding of the softened and enfeebled walls under the *excentric* pressure of the contained blood, now takes the place of hypertrophy, and vascular engorgement of the lungs, distention and dilatation of the right side of the heart, general venous congestion, and anasarca, follow in rapid succession.

In the progress of such a case, therefore, the occurrence of diastolic murmur at the right base would mark an era of great importance, as that of the "turn" of the case, after which general and rapid declension might be predicted.

\* On the subject of the loss of elasticity in yellow fibrous tissue, under long continued or oft repeated tension, the reader is referred to a paper by the author in *The Dublin Quarterly Journal of Medicine*, 1861.

Cruveillier is likewise of opinion that hypertrophy of the left ventricle may cause dilatation and calcification, and even aneurism of the aorta.\*

Amongst the alleged consequences of hypertrophy of the heart are *cerebral* and *pulmonary apoplexy*.

Corvisart, whilst theoretically admitting the occurrence of apoplexy by rupture of the cerebral arteries, either directly from the force of contraction of the left ventricle or indirectly from venous obstruction and reflux upon the arteries of the brain, declares that he cannot recall an instance in which "apoplexy was the evident consequence of a disease of the heart."† Cerebral venous congestion, however, he has repeatedly witnessed.

Bertin, as already stated, regarded hypertrophy of the ventricles of the heart as the immediate cause of active congestion and apoplexy of the brain and lungs.‡

Rokitansky admits an intimate connexion between simple and excentric hypertrophy of the left ventricle, and cerebral apoplexy; but he regards a friable state of the arteries of the brain as a necessary concomitant condition.§ He denies the alleged causal relationship of right ventricular hypertrophy to pulmonary apoplexy.

Hope and Bouillaud regard the connexion between hypertrophy of the left ventricle and cerebral apoplexy, as direct and intimate; but, with Morgagni, they both considered it probable that in all such cases, disease of the arteries of the brain likewise exists. Hope, however, considers hypertrophy the principal factor. But out of four fatal cases of apoplexy under the age of forty years given by him,|| not one presented disease of the heart; whilst, after that age, disease of the heart existed in twenty-five out of thirty-five fatal cases.

Andral is likewise a believer in the direct connexion of left ventricular hypertrophy with cerebral hæmorrhage; but, on the other hand, Fuller regards it as due entirely to the frequent

\* *Anat. Pathologique*, l. xvii., pl. iv., figs. 1 and 2.

† *Opus citat.*, p. 164.

‡ *Maladies du Cœur*, p. 351.

§ *Pathological Anatomy*, vol. i., p. 169.

|| *Opus citat.*, p. 256.

association of atheromatous change in the arteries, with hypertrophy of the left ventricle.\*

In regard to the alleged dependence of pulmonary apoplexy upon hypertrophy of the right ventricle, Rokitansky, as already shown, opposes this doctrine. Hasse and Stokes, on the other hand, uphold it. I believe, however, that derangement of the natural adjustment subsisting between the propulsive force of either ventricle, and the power of resistance of the corresponding arteries, can lead to rupture of the latter only by becoming the cause of disease of the arterial coats, as previously explained (p. 514); and that the occurrence of apoplexy by extravasation, in connexion with ventricular hypertrophy, implies unsoundness of the vascular walls. In the brain the arteries are exposed to a further but extrinsic source of weakness, from softening of the cerebral substance by which, in the normal state, they are supported, and, thereby, enabled to resist high pressure.

The connexion would seem to stand thus. Arterial disease is more common after forty; and the cerebral arteries, being destitute of a *tunica adventitia*, and inadequately supported by brain substance, readily give way when diseased, especially under the impulse of an hypertrophied left ventricle. But hypertrophy of the left ventricle is usually associated with arterial degeneration, either as the cause, or as a concomitant change dependent upon the same morbid agency; *e.g.*, chronic renal disease. Hence, apoplexy would seem to be the product of two concurrent causes; the one predisposing, *viz.*, cerebral arterial disease, and the other immediate, left ventricular hypertrophy. Arterial disease is quite as often the antecedent condition and the cause of hypertrophy, as it is posterior in time, and dependent, however remotely, upon hypertrophy. If hypertrophy in the simple form be more likely than any other to be followed by apoplexy, as Hope surmises, it is because this is the form in which hypertrophy is oftenest found in conjunction with chronic renal disease in its early stages, when the twofold condition of strong propulsive power in the left ventricle, and atrophic changes in the arterial coats, exists.

Doctor Walshe † discusses this question with great acumen,

\* *Diseases of the Chest*, 1862, p. 579.

† *The Diseases of the Heart*, third edition, p. 290.



and arrives at the conclusion that the percentage of cerebral apoplexy, in those affected with hypertrophy of the left ventricle, is but slightly in excess of its ratio where no cardiac disease exists. If the further consideration, that hypertrophy in the great majority of cases is a condition of compensation, be allowed its legitimate weight, and that, with the two exceptions of renal hypertrophy, and hypertrophy consecutive to primary arterial disease, the obstruction to which it owes its origin is located on the cardiac side of the cerebral arteries, the conclusion will seem inevitable, that in hypertrophy of these two forms only, can cerebral apoplexy be regarded as even remotely dependent upon increased propulsive power in the left ventricle.

Whilst denying the deleterious influence of left ventricular hypertrophy on the cerebral circulation, Dr. Walshe is disposed to regard tricuspid inadequacy as a direct cause of cerebral venous congestion. But apoplexy from this cause is exceedingly rare.

*Pulmonary apoplexy*, like cerebral, was, till recently, regarded as the direct result of increased contractile force in the corresponding ventricle. This accident, for such it is, most frequently occurs in connexion with mitral obstruction or inadequacy; but, inasmuch as either of these conditions of the mitral opening is prone to react upon the right ventricle by causing hypertrophy of its walls, the latter change, having usually been accomplished before the occurrence of pulmonary apoplexy, came to be regarded as its sole cause.

This theory, however, I believe to be ill founded. In four only of the thirteen cases of mitral narrowing in which I examined the body after death, was pulmonary apoplexy found to be present (cases of Anne K., Jane M., Jane Q., and Thomas W.). In two of these the hypertrophy of the right ventricle was very slight; in the third no note was made of the state of that chamber; whilst in the fourth the right ventricle was dilated and actually thinned. Conversely, in two cases (cases of R. M'B. and T. N.), right ventricular hypertrophy existed in a high degree; and in one of these (the former) it was extreme; yet pulmonary apoplexy had not occurred.

From the data supplied by my own statistics, I therefore feel

warranted in concluding that no necessary connexion exists between pulmonary apoplexy and hypertrophy of the right ventricle. But whilst, in four only out of thirteen cases of mitral narrowing, or a fraction less than one-third of the whole number, pulmonary apoplexy was found to have occurred, hæmoptysis had actually taken place in eleven, and, to all appearance, irrespectively of the condition of the right ventricle.

In relation to another subject, which has been recently engaging the attention of experimental pathologists, namely, the alleged connexion between pulmonary infarctus and extravasation, on the one hand, and thrombosis of the pulmonary artery and right ventricle upon the other, my statistics afford some useful information.

It would seem from the experiments and observations of Lefevre,\* that obstruction of the artery of supply to a district of tissue, will cause immediate engorgement of the capillaries and general turgescence of the part. According to Feltz† this is due to reflux from the veins and paralysis of the capillaries, and is frequently attended with extravasation of blood. In pulmonary apoplexy, therefore, and in hæmoptysis of cardiac origin, thrombosis of the pulmonary artery or its branches, or of the right ventricle, might reasonably be looked for. In all four cases of mitral narrowing in which pulmonary apoplexy was found, thrombosis of the right ventricle and trunk of the pulmonary artery existed. In two cases of pulmonary apoplexy in connexion with mitral regurgitation, in which the valves were found inadequate, without stenosis of the orifice, right ventricular and arterial thrombosis likewise existed. On the other hand, in several examples of thrombosis of the right ventricle and pulmonary artery, which have come under my notice, pulmonary apoplexy did not exist. It would seem, therefore, that whilst pulmonary apoplexy, whether associated with obstruction or valvular inadequacy at the mitral orifice, is necessarily connected with, if not dependent upon, thrombosis of the right ventricle and pulmonary artery, the latter condition, in the absence of mitral lesion, may exist without pulmonary apoplexy,

\* *British and Foreign Medico-Chirurgical Review*, October, 1871.

† *Loco citat.*

or, in other words, it does not of necessity lead to this complication.

The connexion between hæmoptysis and right cardiac thrombosis would appear to be less intimate. Thus, hæmoptysis had occurred in eleven out of thirteen fatal cases of contracted mitral opening; whilst of these, thrombosis of the right chambers was found only in nine; and in only five out of eight cases of simple mitral inadequacy hæmoptysis had occurred, whilst right ventricular thrombosis existed in seven.

Hæmoptysis was, therefore, in numerical excess of thrombosis in mitral narrowing; whilst the reverse was the case in mitral inadequacy.

From the preceding abstract, the inference may be legitimately drawn, that the connexion of pulmonary apoplexy, and of hæmoptysis, with thrombosis of the right side of the heart, is not the same; pulmonary apoplexy seeming to require it as a necessary antecedent condition; whilst hæmoptysis, though generally associated with thrombosis in the last moments of life, as a further consequence of capillary infarction, may, and frequently does occur independently of it at an earlier period of the fatal illness, and as a direct consequence of the primary cardiac lesion. It may be further inferred, that as thrombosis of the right side of the heart and pulmonary artery is a necessarily and proximately fatal occurrence, apoplexy of the lung, as a consequence of it, is an event of the last few hours of life.

*Fatty degeneration* is a change consecutive to hypertrophy, rather than a consequence of it.

The relative inadequacy of cardiac nutrition, by which hypertrophy of the heart, when it exceeds a certain limit, is necessarily followed, added to the absolute impairment of nutrition which results from dilatation of the aorta, and atheromatous transformation and rigidity of the coronary arteries, at a later period leads to fatty degeneration and failure of the heart. The advent of this condition, which will be discussed in detail further on, is announced by weakness and irregularity of pulse, occasional vertigo, or even partial syncope; on suddenly assuming the erect posture, or making ordinary exertion; frequently, but not always, by tortuosity and rigidity of the superficial

arteries ; and by weak impulse and irregular action of the heart as to rhythm and force ; the sounds at the apex being weak, and when not associated with murmur, either muffled or sharp, according to the thickness of the walls of the left ventricle. At the base the first sound is usually of the same character as at the apex, but it may be clearer ; whilst the second sound, owing to dilatation of the aorta, or atheromatous change of its walls, is invariably sharp and clear when not masked by murmur.

The ultimate consequences of dilatation and debility of the ventricles soon make their appearance ; viz., engorgement of the lungs, and of the right side of the heart and liver, congestive bronchitis, general venous distention, anasarca, diminished secretion of urine with albuminuria, and, finally, effusion into the serous cavities.

The muscular structure of the heart may be found to present the condition of “granular” degeneration, fatty transformation, or “obsolescence” of the fibres ; and frequently all three conditions or degrees of tissue-degradation are met with in the same organ or chamber.

*Gangrene* of distant portions of the body is never a result of hypertrophy of the heart, as such ; but it not unfrequently arises from *accidents* of hypertrophy, namely, thrombosis, or embolism. Inasmuch as these formidable complications of cardiac and vascular disease will form the subject of a special chapter, it is unnecessary to allude to them further in this place.

Is hypertrophy *curable* ? I have already stated that in the absence of its complications, causal and consequential, hypertrophy of the heart should not, in my judgment, be regarded as a disease ; and that, even when associated with disease by dependence or by cause, it constitutes, in the proper sense, no essential portion of the morbid condition, but is rather the product of an effort of compensation, a *nisus naturæ*, to supplement inadequacy of propulsive power in the heart. It constitutes, however, a border condition of proclivity to disease, leading necessarily, in time, to degeneration of the heart, and inviting acute inflammation of other organs, especially the lungs ; which, when established, it most seriously aggravates. Hypertrophy of the heart, being in most cases secondary to organic and incurable



disease, is itself irremovable, save in the comparatively few examples where it is primary; and even here it is only partially remediable, because the calibre and thickness of the arteries have become adapted by usage to the exaggerated propulsion of the ventricles, and are not alterable by treatment.

Hypertrophy is, therefore, not strictly curable in any of its forms; and even were it removable by treatment, to effect its cure would be manifestly undesirable, in view of what has been just stated. Yet Laennec maintained that hypertrophy, whether simple or dilated, was the most curable of all the organic diseases of the heart.\*

Hope was of opinion that in "nearly" all cases where the patient is under forty years old, of good constitution, and free from valvular disease, obstruction of the aorta, adhesion of the pericardium, and softening of the heart, hypertrophy "may be radically cured."† No doubt, under the conditions mentioned, hypertrophy would be found most amenable to treatment directed to its alleviation, and to the arrest of its progress; but even here it is not "radically" curable.

Hypertrophy in the primary form, or that which results from over active and long continued physical exertion, or from habitual emotional excitement, is alone even partially remediable. Nevertheless, the accidents and the consequences of hypertrophy, by whatever cause produced, are only in a limited degree under the control of medicine.

Corvisart approved of the "depletory" system of Valsalva and Albertini, which consisted in repeated bleedings and low diet, as applied to "active aneurism;" but for other forms of the affection he considered it unsuitable.‡ He regarded Morgagni's plan of diverting blood from the heart by plunging the extremities repeatedly into hot water, although useful in alleviating the paroxysms of dyspnoea occasionally witnessed in the progress of hypertrophy, as of only temporary effect in the treatment of the affection itself. Corvisart, nevertheless, advises moderate abstraction of blood in the treatment of all forms of hypertrophy. I

\* *Auscultation Médiate*, tom. ii., p. 732.

† *Opus citat.*, p. 289.

‡ *Opus citat.*, p. 141.

cannot help thinking that the reduction in the rate and force of the pulse under Valsalva's treatment, was misinterpreted by the physicians of that time, unacquainted as they were with auscultation and percussion, as evidence of decrease in the volume of the heart.

This remark will certainly not apply to the illustrious Laennec, nor indeed to his scarcely less distinguished compatriots and contemporaries, Bertin and Bouillaud; all of whom give an unqualified sanction to Valsalva's treatment; but with grave misgivings that patients will be found to possess the courage and perseverance to submit to it for months, and even, with brief interruptions, for years, as Laennec suggests may be necessary.

Laennec ventured to promise a large measure of success from this plan of treatment, if it be applied early, and before the grave general effects of hypertrophy have supervened. He commenced with a general bleeding to the verge of syncope, and repeated this every two, four, or eight hours; or less frequently, according to the effect, till palpitation ceased, and the impulse was reduced in force. The food should be, at the same time, reduced to one half the usual allowance; and even still further if the patient's strength exceed what is barely necessary for a few minutes' walk. For a vigorous adult, his ordinary allowance was fourteen ounces of solid food per day, two ounces of which consisted of white meats; and if soup or milk was taken, four ounces of either counted as one of solids. No wine was allowed. If, at the end of two months, palpitation and strong impulse no longer existed, "the intervals between the bleedings might be prolonged, and the severity of the regimen somewhat relaxed."\* Not even the existence of dropsical effusion would deter him from the practice of this heroic treatment. In addition, he prescribed diuretics and drastic purgatives.

Bouillaud, in the treatment of moderate hypertrophy in a man of medium strength and appetite, would practice three or four bleedings from the arm, each amounting to three or four *palettes*, to which he would add in the course of treatment, one or two cuppings, amounting to two or three *palettes*. He would also prescribe digitalis, which he designates the "veri-

\* *Traité de l'Auscultation Médiate*, tom. ii., p. 733-4.

table opium of the heart." This latter medicine he uses by dusting the blistered surface of the precordium with six to fifteen grains of the powder daily ; being of opinion that it acts as a local sedative.\*

The practice of these two eminent men, thus given in summary, will suffice to illustrate the authorized treatment of hypertrophy of the heart at the period when they wrote. Yet who would be bold enough to pursue it, even in a mitigated form, now ?

Hope's plan of treatment might be represented as the preceding, but in a less heroic form. He recommended that two to eight ounces of blood should be drawn every two to six weeks, according to the age and strength of the patient. If there had been threatening of apoplexy, the preceding quantity of blood should be taken, by means of cupping glasses, from the nape of the neck. The diet should be regulated and restricted, especially for the plethoric ; and it should consist, for the first few months, of white fish and farinaceous and vegetable food. Drink should be restricted, and alcoholic stimulants strictly prohibited. Diuretics and hydragogue purgatives should be administered, especially in presence of dropsical effusion. If the patient were anæmic or feeble, iron and tonics might be necessary ; and in case nervous palpitation existed, sedatives, such as opium, hyoscyamus, and digitalis, would be demanded. Under this plan of treatment, continued from one to two years, he declared that his recoveries were numerous.†

Walshe limits blood-letting in hypertrophy to the application of four or five leeches over the heart, having very properly the fear of anæmia before his mind. He especially recommends aconite as a tranquillizing agent.‡

I believe Dr. Latham was the first writer who pointed out the evil effect of the active depletory treatment of hypertrophy ; especially of general and repeated blood-letting. He showed that the state of anæmia thereby induced, by rendering the heart morbidly susceptible of ordinary impressions, more than neutralized

\* *Traité Clinique des Maladies du Cœur*, 1835, tom. ii., p. 460.

† *A Treatise on the Diseases of the Heart and Great Vessels*, 1839, p. 284-9.

‡ *Ibid.*, fourth edition, 1873, p. 297-8.

the tranquillizing action of the measures employed; and by inducing debility, favoured the occurrence of dilatation of the heart.\*

In discussing the treatment of the consecutive hypertrophy of aortic valve inadequacy, Sir Dominic Corrigan† incidentally and ably exposes the dangers resulting from the active depletory system of treatment of all forms of hypertrophy.

Doctor Stokes no less strongly deprecates this plan of treatment, as likely to lead to the twofold evil result of softening and dilatation.‡

The two last named physicians recommend the occasional application of a few leeches to the precordium, and the avoidance of all causes of excitement of the heart. If the patient complain of constriction of the chest, with oppression and dyspnoea, and exhibit at the same time a full and bounding pulse, Sir D. Corrigan advises a moderate venesection. Dr. Stokes recommends the use of mercury where the liver is engorged in a gouty subject. He likewise advocates the use of digitalis, in doses of ℞x-xv of the tincture thrice daily, as a tranquillizing agent in the nervous palpitation of hypertrophy.

Recent experimental inquiry into the action of digitalis upon the heart, and, indeed, my own experience of the drug in the treatment of cardiac disease, would not sanction this use of it. It is essentially a cardiac tonic and stimulant, imparting power to the heart, and regularity to its action. In the treatment of hypertrophy, therefore, it is admissible only where weakness and irregularity exist; namely, in the condition of softening and dilatation.

The treatment of *primary* hypertrophy, whether due to physical or moral agency, should consist, in the first instance, in the adoption of measures against continued or repeated excitement of the heart. Long or rapid walks, or over active exercise of any kind, should be accordingly avoided; as likewise the use of alcoholic stimulants, and mental or emotional excitement. Nevertheless, open air exercise, even of an active kind, but within the limits of moderately quickened circulation, should be taken,

\* *Lectures*, No. xxxi.

† *Edinburgh Medical and Surgical Journal*, vol. xxxvii., 1832.

‡ *The Diseases of the Heart and the Aorta*, 1854, p. 346.



in order to prevent derangement of primary digestion, and to promote renovation of tissue. Whilst active measures, designed to reduce the volume of an already enlarged heart, should be studiously avoided, as calculated to lead to degeneration of its structure, paroxysmal excitement or palpitation should be controlled by the application of a few leeches to the precordium, if a feeling of oppression, and some difficulty of breathing, with or without headache, and a full and strong pulse, be associated with the palpitation. But if the cardiac excitement be chiefly nervous, as indicated by a pallid and anxious face, with dilated pupils, quick, but not strong pulse, and a thrilling vibration communicated to the hand placed over the heart, then the proper remedies would be morphia with hydrocyanic acid: ℥x-xv of the solution of the former, acetate or hydrochlorate, with ℥ij-v of the latter, of Pharmacopœial strength, in half a wineglassful of water, given every hour until excitement be subdued and relief obtained. *Veratrum viride* may be likewise prescribed with advantage in similar cases, from ℥v-x of the tincture in spearmint water every second hour, till the desired effect is produced, its action being carefully watched.\* Better than either of these is the etherial tincture of Indian hemp, in doses of ℥x-xxx suspended in water by means of mucilage, and given every second hour, if necessary, under careful supervision. Aconite may be likewise given in similar cases with good effect; it should be administered in doses of ℥v of the Pharmacopœial tincture every third hour, until the rate of pulsation subside. In sthenic palpitation, the bromides of ammonium and potassium may be given singly or combined, in gr. x doses, at intervals of a few hours, with the best results; but if cardiac debility exist, I would not prescribe these salts. *Digitalis* would be the remedy in case of palpitation, characterized by feeble impulse, with irregular action of the heart. It should be given in doses of ℥x-xx of the tincture, combined with ℥v of spirit of chloroform every third hour, during the continuance of cardiac excitement. The administration of a full enema, with oil and spirits

\* Bullock has recently obtained from this agent two alkaloids, *viridia* and *veratroidia*, which are eminently depressant of the heart's action, both in regard to force and rate, and may be administered subcutaneously.

of turpentine, often materially aids in relieving palpitation when the bowels are confined and inflated. The diet should be nutritious and non-stimulating, containing a large percentage of nitrogenous aliment, and a diminished proportion of fat and hydro-carbons generally; the object to be held in view in prescribing regimen and diet being, not a reduction in the volume of the heart, but the maintenance of it in a state of structural integrity, and the avoidance of fatty change. I have not tried acetate of lead in the treatment of hypertrophy, with the object of reducing the volume of the heart; nor, indeed, would I expect satisfactory results from its use.

The treatment of hypertrophy in the consecutive form will depend upon the particular cause to which it owes its origin. Thus, hypertrophy of renal origin requires for its treatment measures directed to the disease of the kidneys almost exclusively. When, however, in the latter stages of the affection, the left ventricle, by deterioration of its structure, begins to yield under pressure from within, symptoms indicative of over-repletion of the left side of the heart, passive congestion of the lungs, and engorgement of the right chambers are exhibited. A feeling of pectoral constriction with dyspnoea is now experienced; the breathing is quickened and less deep; the carotids throb forcibly; the great veins of the neck are engorged, and the pulse is quick, slender, and irregular. Under these circumstances, iron and digitalis, or the triple compound of iron, strychnia, and quinine, is especially indicated. If the evidence of dilatation and distention of the chambers be in excess of that of muscular and neurotic failure of the heart, digitalis is the appropriate remedy. I usually prescribe ℞-xv of the tincture, with an equal quantity of the tincture of perchloride of iron, and of chloric ether, or spirit of nitrous ether, every three or four hours. The latter is generally preferred for its diuretic action. Where, however, the pulse is feeble and irregular, without characteristic respiratory distress or venous congestion, the heart's action being rapid and weak, with unequal and abortive contractions of the left ventricle, not represented in the radial pulse, occurring from time to time, strychnia and quinine should be preferred, and are best given in combination with iron in small doses. Thus:

R. *Liquor strychniæ*, *Acid. sulphur. dil.*, aa. ℥ xxx; *Syrupi limon.*, ʒiv; *Sulphatis quiniæ*, gr. xvj; *Sulphatis ferri granulat.*, gr. iv; *Aquæ font.*, q.s. ad ʒviij. Half an ounce to be taken every third hour.

Half an ounce of whiskey or brandy diluted with water, or half a pint of good bitter beer, should be likewise allowed twice or thrice daily; because, in addition to their stimulant effect upon the heart, they possess decidedly diuretic properties. Where engorgement of the superficial veins exists, there is generally, likewise, congestion of the kidneys and liver. Hence cupping of the loins, with or without abstraction of blood, and followed by the application of a linseed poultice, would be advisable as a means of promoting the secretion of urine, and thereby reducing cardiac and vascular pressure. With a similar object in view, drink of any kind should be allowed only in very small quantity, but often if necessary; and, as a means of unloading the vessels generally, and those of the liver in particular, saline aperients and resin of podophylline, the latter in gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  doses, with compound colocynth or aloetic pill, should be administered from time to time.

The treatment of hypertrophy dependent upon valvular disease, resolves itself into that of the accidents of the valvular lesion present, and of the attendant dilatation. From the strain occasionally put upon it by injudicious exercise or muscular effort, a heart the subject of valvular disease and hypertrophy, with dilatation or fatty metamorphosis, may act inordinately.

The palpitation thus produced is best treated by the application of two or three leeches over the base of the heart, followed by a warm cataplasm, a moderate opiate being given immediately afterwards. An opiate plaster should be subsequently applied over the heart as a mechanical support and sedative. The intercurrent symptoms due to dilatation have been already discussed; the treatment then sketched, which it is unnecessary to recapitulate here, is equally applicable in the circumstances now under consideration.

The following cases will serve to illustrate the various forms of hypertrophy described in the preceding pages, and the treatment applicable to each form of the affection.

CASE XXI.—*Albuminuria ; General Dropsy ; Orthopnœa ; strong Impulse of Heart ; Hæmatemesis ; Sudden Death. Cirrhosis of the Liver ; Fatty Degeneration of the Kidneys ; “ Concentric ” Hypertrophy and Granular Degeneration of the Heart.*

John O'D., aged forty-four years, a discharged soldier of intemperate habits, who had served several years in India and other warm climates, was admitted January 8th, 1866. Four months previously his attention was arrested by frequent calls to pass water. A month subsequently he noticed a swelling of the ankles, and, shortly afterwards, of the lower extremities generally, and of the abdomen. When admitted, he was generally dropsical. The lower extremities and genitals were very much distended ; there was likewise ascites and flatulent distention of the bowels. Ramifying over the left side of the abdomen were several large, tortuous, and distended veins, the colour of which contrasted strongly with that of the skin, which was of waxy whiteness. The face, chest, and arms were likewise œdematous. Respiration was much embarrassed, and the pulse was quick, but regular, and rather strong. The heart pulsated with considerable force in the usual situation ; its sounds and rhythm were normal ; but precordial dulness was extended. There was orthopnœa. The urine was highly albuminous, sp. gr. 1010 ; reaction acid, and under the microscope the precipitate exhibited hyaline tube casts, some simply destitute of epithelium, and others studded with fat granules. Respiration was loud on both sides, except posteriorly and inferiorly on the right, where it was feeble and accompanied with râles. Here, likewise, was percussion-dulness ; but elsewhere there was normal resonance. There was manifestly renal disease. That the liver was likewise the subject of disease, probably cirrhosis, was inferred from the great disproportion between the ascites and the dropsical effusion elsewhere ; from the distended state of the superficial abdominal veins, showing visceral obstruction beneath ; and from the previous intemperate habits of the patient. Finally, hypertrophy of the heart was diagnosed from the abnormal extent of precordial dulness, and the strong impulse at the precordium. Elaterium was tried ; but the stomach revolted against it.



On the night of the 10th he vomited a large quantity of dark blood; and on the following morning was found breathing stertorously, the pulse irregular and intermittent, and the impulse of the heart imperceptible. Effusion was manifestly taking place into all the serous cavities, including the pericardium. He was quite conscious. In the attempt to sit up in bed for the purpose of describing his sensations, he suddenly died in a state of syncope.

The peritoneal cavity was found distended with serum; the liver in a most advanced stage of cirrhosis; and the spleen enlarged, its capsule thickened and adherent to the diaphragm, and its artery tortuous, and nearly as large as the arteria innominata. The left kidney was somewhat enlarged, and, on section, presented a mottled appearance. The cortex was rather increased in thickness, and examined microscopically it showed fatty disease in an advanced stage. The pyramids were enlarged. The right kidney resembled the left in general appearance, but was much smaller, scarcely exceeding in size the suprarenal capsule of the foetus.

The pericardium was full of serum. The heart was hypertrophied on the left side, rather pale in colour, and loaded with superficial fat. The right cavities and the tricuspid orifice were dilated, the latter admitting four fingers to pass through it with facility; the walls of the right ventricle were much attenuated. The left auricle was rather thickened, and the left ventricle "concentrically" hypertrophied. The papillary muscles were likewise thickened. The cavity of the left ventricle was reduced in capacity. The valves were everywhere healthy.

The pleural cavities were full of serum. The right lung sank in water, but admitted of inflation; the left lung floated.

The following measurements were taken five days subsequent to the patient's death, and after much and forcible stretching of the ventricular walls:

Heart	...	Circumference at base of ventricles, $11\frac{3}{8}$ ".
Aorta	...	Circumference, two inches above valves, $3\frac{1}{16}$ ".
Left ventricle ...	{	Wall, at apex, $\frac{7}{8}$ " thick.
		,, middle, $1\frac{1}{8}$ " ,,
		,, base, $1\frac{1}{3}$ " ,,
		Cavity, length from aortic ring to apex, 3".
		,, width from middle of septum to opposite wall, $1\frac{1}{2}$ ".
		Circumference at base, $10\frac{1}{8}$ ".

Left auricle ... Wall in central portion,  $\frac{1}{8}$ " thick.

Right ventricle { Wall, at apex,  $\frac{1}{8}$ " thick.  
 ,, middle,  $\frac{1}{4}$ " "  
 ,, at base,  $\frac{1}{8}$ "

A section of the wall of the left ventricle, examined under the microscope, exhibited a "granular" condition of the muscular fibre.

Hypertrophy of the left ventricle in this case was, in my judgment, consecutive to renal and hepatic disease, and "simple" in the first instance. Granular degeneration of the substance of the heart next took place; and subsequently the right ventricle, weakened by this change, underwent dilatation under the increased blood-pressure caused by pleural effusion and obstructed pulmonary circulation. The left ventricle, already hypertrophied, was "surprised in the full vigour of contractility" by failure of the right ventricle and stoppage of the circulation, in the effort of the patient to sit up.

The state of "concentric" hypertrophy I therefore regard as consecutive, in this instance, to the "simple" form; and as a product, not of a pathological process, but of the mode of dissolution.

CASE XXII.—*Albuminuria; General Dropsy; Diarrhœa; Jaundice; Erysipelas, and Gangrene; Hæmoptysis; Death. Fatty Disease of the Kidneys, Liver, and Spleen; Congestion of the Lungs, and Pulmonary Apoplexy; Hypertrophy and Dilatation of the Left Ventricle; Dilatation of the Right Ventricle; Advanced Fatty Degeneration of the Heart.*

Thomas M'K., aged forty-four years, coachman; of temperate habits; admitted May 5th, 1866. Two months previously, up to which period his health was excellent, he caught cold from exposure after working in the heated atmosphere of a conservatory, and was troubled with cough.

On admittance, he suffered from great dyspnœa; there was œdema of the face and feet, and the heart's action was laboured, and felt at the scrobiculus cordis. Pulmonary resonance was exaggerated; and respiration was feeble and accompanied with râles all over the chest; expectoration copious and frothy; urine, sp. gr. 1.020 and albuminous. Diagnosis: pulmonary emphysema with

bronchitis, and engorgement of the right heart and kidneys. The treatment consisted in blisters to the chest; stimulant expectorants, including tincture of larch bark and turpentine punch;\* and, subsequently, quinine, and good diet. Discharged, May 23rd, so much improved as to be able to resume his work.

Readmitted on the 26th June. There was then much cedema of feet and legs, and also slight puffiness of face. Respiration was embarrassed; urine, sp. gr. 1.015, loaded with albumen, and, of late, occasionally likewise with lithates. After a few days the scrotum and penis became swollen, and required puncturing. About half an ounce of clear serum, collected from the punctures, was tested by ebullition the following day and found to contain only a trace of albumen. Astringents were employed to check diarrhœa which was occasionally present; blisters were applied to the chest; turpentine punch was administered; and, subsequently, iodide of potassium in infusion of cinchona.

July 12th. Pulse 114, and barely to be felt; respiration 66. Both sides of the chest were dull half way up, and to the same extent respiratory sounds were absent; sputa rust-coloured and viscid; lower limbs and genitals swollen. The chest was wrapped in a warm poultice, and whiskey punch was given.

July 17th. Pulse 114; respiration 42. Patient remarked that the rate of his respiration sometimes varied; ascites; genitals much swollen, and prepuce punctured. Baillie's pills were given twice daily.

July 21st. Has been taking tinct. digitalis in  $\mathcal{M}$  x doses, with spirit of juniper, for the last two days, notwithstanding which there is all but complete suppression of urine. Œdema has increased, and now extends to hands. Pulse scarcely to be felt, and not to be counted at the wrist. To have grs. xv of compound powder of jalap, with gr.  $\frac{1}{12}$  of elaterium twice daily.

July 23rd. Patient is deeply jaundiced. Elaterium has acted twice, and penis is less swollen. The entire of the left lower extremity, to the hip, is in a state of erysipelatous inflammation, threatening gangrene. Lead lotion was directed to be kept constantly applied to the inflamed surface.

\* Known in Dublin as "Parr's Punch," and made with whiskey and spirits of turpentine, in equal proportions, diluted with sweetened hot water to the proper strength.

July 25th. The entire of left lower extremity is in a state of gangrene. From the knee downwards the limb is studded with large bullæ, and the cuticle detached. The thigh is red and tense; and inflammation has extended to the scrotum, which, on each side, is passing into a state of gangrene. Patient all but pulseless; but is quite conscious and free from suffering; face deeply jaundiced and shrunken.

July 26th. Entire left leg and thigh dark and sloughy; scrotum likewise in a state of gangrene, which extends in dark streaks over the front and upper portion of the right thigh. Respiration tranquil, and patient still hopeful.

28th. Spat up a good deal of blood last night, and died at half past nine o'clock this morning.

*Post mortem* examination seven hours afterwards. There was copious effusion into all the serous cavities. Kidneys large, fatty, and smooth on the surface; the cortex was much thickened, and of a light yellow colour; minute oil granules, as exhibited under the microscope, were interposed between the tubes and blood vessels; the epithelium and gland structure, however, seemed healthy. The liver was large, yellow, and readily torn; and, examined microscopically, the hepatic cells in most instances were found to contain eight to ten highly refracting oil spherules; large oil drops were likewise dispersed over the field of vision. Spleen fatty, and fissured.

Lungs healthy structurally; but bases were congested and solid, and smooth dark and dry in section; they floated in water. In the anterior edge of the right lung was a nodule of blood as large as a bantam's egg; in a corresponding situation in the upper lobe of the left lung, and also in the upper portion of its inferior lobe, were similar but larger collections of extravasated blood.

The heart was large, and, with one inch of the aorta and pulmonary artery attached, weighed fourteen and a-half ounces. The right cavities contained some dark blood; their walls were soft and thinned. Left cavities much dilated, and walls slightly thickened. Valves all absolutely healthy. Aorta of ordinary size, and its coats healthy. Heart flabby, and, when laid on the table, was flattened out by its own weight. On section the walls



presented a yellow hue, and a portion examined microscopically presented a good example of fatty degeneration of the muscular substance, the fibres consisting of linear rows of minute circular refractile dots about 10,000th part of an inch in diameter. These were likewise abundantly scattered in a free state over the field, as if by liberation from the sarcolemma.\* The outline of the fibres was quite distinct; but, with the exception of a few places where it was very faintly visible, transverse striation was entirely obliterated.

From the outset, I regarded this case as an example of primary disease of the kidneys, with consequent hypertrophy and consecutive fatty disease of the heart. It is probable, however, that fatty change of the heart and kidneys was cotemporaneous: the former supervening upon a state of simple hypertrophy, and the latter upon one of sub-inflammatory or congestive enlargement. The liver and spleen would seem to have undergone the fatty transformation at a later period, under the influence of the general impairment or perversion of nutrition. Pulmonary apoplexy and hæmoptysis were probably the result of mitral regurgitation by yielding of the walls of the left ventricle, dilatation of its cavity, and consequent reflux by inadequacy of healthy valves, shortly before death. The jaundicing is of great diagnostic value in such cases, as serving to distinguish structural disease of the liver from simple congestion.

*CASE XXIII.—Oppression and Palpitation supervening on emotional shock; Paroxysmal Dyspnœa; Hypertrophy with Dilatation; Death. Fatty Degeneration of the Heart; Dilatation of the Aorta.*

Peter S., a gentleman farmer from the county of Meath, aged fifty-six years, was admitted into hospital as a private patient, May 30th, 1866. Seventeen months previously, after some severe family afflictions, he experienced rather suddenly, and for the first time, oppression of the chest and palpitation, accompanied by nervousness and great anxiety. Since that date he has been subject to occasional attacks of dyspnœa with restlessness and anxiety. Has likewise had from time to time a feel-

\* I am convinced that the muscular fibres of the heart possess a sarcolemma.

ing of great debility and sinking, not amounting, however, to syncope.

Is rather fresh in colour; pupils dilated, and face expressive of fear and anxiety; slight œdema of feet and legs; pulse 104, and remarkably weak but regular. Chest universally resonant. On right side respiration is feeble, and accompanied in supra-mammary region with slight crepitant râles; on left side it is puerile.

Precordial dulness extended; impulse of heart strong at apex, and here the first sound is obscure. Over the base and at mid-sternum, there is a murmur with, and masking, the second sound; but it is not transmitted more than an inch above the aortic orifice. The carotids pulsate visibly, but not the temporal or radial arteries. Fauces congested. No appetite for food. Orthopnea. Sleep disturbed by unpleasant dreams and startings.

Diagnosis: dilatation of the aorta, hypertrophy, and probably fatty degeneration of the heart. To have Hoffman's anodyne and Battley's sedative in moderate doses with camphor water every third hour, and a little brandy and water occasionally. On the 1st of June, after a rather tranquil night, and whilst the patient was under examination, the breathing was noticed to be singularly irregular; it was quick, loud, and suspirious; but immediately the examination of his chest was suspended, it became slow, and was entirely suspended during a period of several seconds. Tongue loaded; stomach and intestines inflated. To have pil. rhei co., gr. x. Stop brandy. He died on June 11th, manifestly from inanition and exhaustion.

*Post mortem* twenty-four hours after death. Much liquid in pericardium. Heart flabby and enlarged, weighing twenty-seven ounces, with an inch of the aorta and pulmonary artery attached. There was a good deal of fat on the surface of the heart; and at the apex, and partially embedded in the muscular substance, there was a solid mass of fat one-eighth of an inch thick.

The anterior surface of the right ventricle presented a large "milk spot." The right chambers were distended with dark coagulated blood; they were greatly dilated, as was likewise the tricuspid orifice. The walls of the right ventricle were somewhat

thinned. The left ventricle was much thickened and dilated. Aorta greatly dilated for some distance above orifice, and its walls slightly atheromatous. Valves all structurally healthy. Microscopically examined, the muscular structure of the heart presented the following appearance. The outline of the fibres was well defined, and the transverse striæ were pretty well seen; but in several of the fibres, and in the direction of their long axes, minute transparent dots were visibly disposed in linear series. In these situations the striæ had disappeared.

There had been total loss of appetite for several days preceding death, the patient absolutely refusing solids of any kind, and taking only an infinitesimal quantity of liquid nutriment in the form of beef-tea with a little brandy. It seemed as if the tissues of the body had ceased to assimilate, long anterior to somatic death. The tongue, during this period, was dry, brown, and crusted.

As to pathological sequence, I incline to the opinion that there was primary atheroma of the aorta, and consequent left ventricular hypertrophy; and that from this twofold cause arose sudden dilatation of the aorta by overstrain, on the occasion of the emotional excitement already mentioned.

The diagnosis of dilated aorta rested mainly upon the phenomenon of paroxysmal dyspnoea, which I regard as pathognomonic of this special condition. But on this subject generally I will state my views more in detail in a future chapter.

CASE XXIV.—*Rheumatism ; Systolic Apex-Murmur ; Death. Hypertrophy of Left Ventricle, and incompetence of Mitral Valve from Rupture of Tendinous Chords; Pulmonary Apoplexy.*

George P., aged thirteen years, admitted November 7th, 1866, under Dr. Hughes, and came under my notice on the 15th whilst I was doing temporary duty for him. The boy had rheumatic fever three months previously, from which he made a satisfactory recovery; but, shortly afterwards caught cold by going out without his boots on a wet day, and has been suffering from cough and violent palpitation since that time.

When first seen by me, his face was bloated and slightly con-

gested, the cervical veins distended, and the external jugulars pulsating synchronously with the heart; supra-clavicular fossæ obliterated; chest barrel-shaped and nearly motionless. Percussion-resonance was exaggerated, and respiration loud over the front of the chest. Cardiac impulse strong, and precordial dullness extended to right side. Fremitus over extreme left apex; second sound sharp and clear, but first sound replaced by a loud bellows-murmur, terminating with a faint, squeaking, musical note, resembling that elicited by blowing through a reed. At the base of the heart, and over the front of the chest generally, both sounds were heard; the second normal, and the first represented by a bellows-murmur similar to, but less loud than, that heard at the apex, and louder in the course of the pulmonary artery than in that of the aorta. The base of the right lung was dull posteriorly, and over the entire posterior surface of the chest a loud systolic murmur was audible. Liver smooth on surface, and extending below level of umbilicus. Respiration quick, shallow, and almost exclusively abdominal; and respiratory movement confined to left side, where it was very active. At base of thorax there was a circular constriction, corresponding to the attachments of the diaphragm and apparently caused by its action. There was dry cough, with occasional expectoration of blood-stained mucus. Pulse 120, weak, and dicrotous. No visible pulsation of arteries. Urine, sp. gr. 1.030, slightly acid, and free from albumen; feet cedematous.

I ceased to have charge of the patient on the 20th November. The entire body became subsequently anasarctous in an extreme degree; and for the last few days of life, the face and neck turgid, and the extremities livid.

Death took place on the 14th of December.

*Post mortem* six hours after death. Both pleural cavities were full of serum; the lungs were engorged but crepitant generally, and in the middle and inferior lobes of the right were two masses of extravasated blood, each the size of an orange. The base of the left lung was solid; it sank in water, and on section was dry and spleen-like, yielding neither blood nor serum. The pericardium contained some ounces of serum. The heart was considerably in excess of the normal size, smooth on the sur-



face, and free from adhesion. The right chambers contained a little dark and partially coagulated blood; the right ventricle was somewhat dilated and thickened, the tricuspid orifice and valves normal, as were likewise those of the pulmonary artery. Fossa ovalis remarkably deep and recessed, and the operculum adherent to the left surface of septum, at some distance from the margin of fossa, which was, however, imperforate, not admitting of direct communication between the auricles. Left auricle normal; left ventricle dilated and thickened, but not in a high degree. Mitral valves structurally healthy; but the free margins of both segments, to the extent of a quarter of an inch at their anterior and left extremities, had been detached from the tendinous chords, the white mamilloid fragments of which, about two lines in length, remained in connexion with the margins of the valves and the adjacent fleshy columns respectively.

Water poured into the left ventricle flowed freely into the left auricle through an aperture, as large as a pea, bounded by the pendulous edges of the valves.

The aorta was reduced in size, barely admitting the index finger from the ventricle. The aortic valves were competent, and both they and the coats of the aorta were perfectly healthy.

There was a good deal of serum in the peritoneum. The liver was large and mottled white, with intervening dark red areolæ; and on section it presented light areolæ, each surrounded by a dark red vascular surface, as in the early stage of portal venous congestion. Kidneys and spleen healthy.

The history of this boy's illness did not date back beyond three months, when he had rheumatism; this, however, left none of the ordinary traces upon the heart. Rupture of the left *chordæ tendineæ* would seem to have occurred subsequently to this attack, when the boy caught cold; as shown by the occurrence of dyspnœa and palpitation then for the first time, yet without the history of strain or other injury to the chest. In this view the case is further remarkable, as exemplifying functional narrowing of the aorta from reduction in the volume of blood circulating through it within the short period of three months.

Strong impulse of the heart, with weak pulse, is diagnostic of stenosis of the aorta. If this arise from lesion at the aortic

orifice, whether valvular or annular, there will be either a single aortic murmur, systolic in rhythm, or a double murmur, both systolic and diastolic, of similar origin. The absence of any such murmur, when strong cardiac impulse and weak pulsation at *both*\* wrists are detected, would warrant the positive diagnosis of functional narrowing of the aorta, such as that presented in the foregoing case. Aortic stenosis of this kind, when not the result of congenital imperfection of the interventricular septum, or of patency of the foramen ovale, is consecutive to mitral stenosis or reflux, and is therefore usually associated with either presystolic or systolic murmur at the left apex.

CASE XXV.—*Double Aortic Murmur ; Dysphagia, with Hæmoptysis ; Death. Hypertrophy and Dilatation of both Ventricles ; Detachment of segment of Aortic Valve ; Double Congestive Pneumonia ; Enlargement of Bronchial and Mediastinal Glands.*

John R., aged thirty years, painter's assistant, admitted November 16th, 1866 ; served as a soldier for some years, and whilst engaged in Africa in defending a stronghold against a raid of the natives, was thrown from a rampart and sustained a shock on the front of the chest. He felt stunned ; but subsequently recovered, and as he believed, perfectly. This accident occurred some years ago. He had been intemperate for twelve months preceding his admittance into hospital ; but had never been seriously ill till six months prior to that date, when he began to get thin, and had nausea and partial loss of appetite.

On the 12th of November, four days before his admission, he experienced what he described as a choking sensation in the neck, and some difficulty of swallowing, accompanied by nausea and vomiting. These symptoms lasted three days, and then all passed away except irritability of stomach, which continued some time longer.

When the man was first seen by me his legs were slightly cedematous ; the veins of the neck were remarkably congested,

\* I have in view here the possibility of embolic infarction of the brachial or radial artery on one side ; occlusion of the subclavian by the pressure of an aneurism or other tumor ; or congenital malposition or dwarfing of the radial.

and especially so on the left side. There was a loud hacking dry cough. Pulse 108, weak, but regular, and equal on both sides. Pulmonary resonance and respiratory sounds were normal over the entire front of the chest; precordial dulness was likewise normal in extent and degree. Posteriorly there was some slight dulness over the bases of both lungs; but otherwise the condition of these organs was normal. A double murmur existed at the base of the heart, systolic and diastolic in time. Of these, the diastolic murmur was relatively louder; it was transmitted through the ascending aorta, but with diminished intensity, and (for a diastolic murmur a very unusual circumstance) likewise into the great arteries of the neck, and diffused over the front of the chest generally. Posteriorly a single murmur was faintly heard at the inferior angle of the left scapula.

On the following day, 17th, I found him perspiring profusely, and complaining of pains in the ankles and knees, which were swollen. Pulse 120.

On the 18th he spat a little blood, the right side of the chest being dull posteriorly; and on the evening of that day he vomited a quantity of bile-stained liquid mixed with blood.

On the 19th the pulse had come down to 102. He was perspiring freely, and complained of pains in the ankles and calves of the legs; a single systolic murmur was audible at the apex of the heart; dulness was increased both in extent and degree over both sides of the chest posteriorly; and, over the bases of both lungs a loud muco-crepitus was heard. The temporal and cervical veins were distended. There was likewise, at this date, visible pulsation or throbbing of the anterior tibial arteries and all their branches even to the toes, accompanied with vibration sensible to the hand.

From the 19th to the 23rd, when he died, no general examination of the chest was made, owing to his extreme restlessness. During that interval he complained of undefined pains in the knees and ankles, was restless, slept but little, and had a loud spasmodic cough with continual expectoration of blood. On the day preceding his death, which took place suddenly on the night of the 23rd, it was remarked that the temporal arteries pulsed strongly.

*Post mortem.* Both pleural cavities were full of straw-coloured serum, and the pericardium contained a pint of similar fluid. The lungs were engorged with blood, but resonant, and floated in water, with the exception of the inferior lobe of the left. The heart was somewhat larger than in health, and globoid in figure. The right cavities were distended with dark and partially coagulated blood, the clot extending from the right ventricle into the pulmonary artery. The right ventricle was slightly dilated, and the tricuspid orifice readily admitted the points of the four fingers and thumb. The left chambers were likewise distended with dark coagulated blood, and the left ventricle was dilated, and its walls were thickened. The mitral orifice and valves were normal; the aortic orifice, viewed from the ventricle, was likewise apparently healthy, but when examined on the arterial aspect it was found that the right anterior segment of the sigmoid valve had been partially detached from the root of the aorta; its left angle, to the extent of nearly half an inch, hanging loosely into the ventricle in the natural position of the heart. Water poured into the aorta at once flowed into the ventricle through the patulous orifice. The valves were otherwise perfectly sound, and entirely free from disease, as was likewise the aorta. The bronchial glands were greatly enlarged and pressed upon the bronchi, and several large glands were found in the posterior mediastinum detruding the oesophagus to the left, and pressing upon the posterior wall of the trachea. These glands were solid, dark in colour, and mottled with white spots on the superficial surface, and likewise on the surface of section. In the centre of one of these glands there was a mass of hard cheese-like matter, which, on pressure, yielded a quantity of pus. On examination, microscopically, the cheesy substance was found to be tubercular.

There can be no doubt, judging from the man's previous history, and from the evidence afforded by the *post mortem* examination, that the rupture of the aortic valve occurred at the time when he was thrown from the rampart. It may be easily supposed that if, at such a moment, the chest was in the acme of expiration, the sternum being thereby brought into close contact with the pericardium, and through it with the root of the aorta, and if, during the diastole of the ventricle and at the moment of



reaction of the aorta upon its contents, the semilunar valves being closed, a severe shock were received upon the sternum, the valve segments might be thereby ruptured or rent from their attachments.

An accident attended with such consequences in a heart previously sound is, however, exceedingly rare; Dr. Peacock having been able to find only seventeen examples recorded. The case is interesting, moreover, as exemplifying the existence of the physical signs of disease of the aortic valve, both obstructive and regurgitant, and dependent entirely upon an accidental lesion in the absence of disease of the heart or valves; and as showing that left scapular murmur, though usually dependent upon obstruction or regurgitation at the mitral orifice, may be caused by lesion of the aortic valve exclusively.

Temporary dysphagia with hæmoptysis may be due to causes other than thoracic aneurism, as this case proves; and, in regard to the pulmonary complication which it presented, Dr. Stokes has well remarked upon the frequent occurrence of pneumonia of a low type in connexion with inadequacy of the aortic valve; as well as upon the frequency with which, under such circumstances, sudden deaths occur.

As to the differential diagnosis between ruptured aortic valve and lesion of the same valve arising from disease: irrespectively of the aid afforded towards forming a correct judgment by the history of the case, the most valuable sign, and one which is in a degree pathognomonic of partial detachment of the valve, is the occurrence of *arterial thrill*, of systolic rhythm, in connexion with double murmur at the aortic orifice. I should henceforth regard such a phenomenon, in all doubtful cases, as conclusively establishing the existence of a loose and vibrating valve-flap at the root of the aorta, where no ground for suspecting thrombosis or embolism existed. Embolism would declare itself by other and indubitable evidence; whilst the precipitation and attachment of a flake of fibrin at the mouth of the vessel would give rise to a thrill of only temporary duration, and would yield a murmur most probably musical in quality. A diastolic basic murmur, audible in the arteries of the neck, does *not* belong to aortic patency from disease; it is a phenomenon so strictly exceptional

that I have never heard it, save in this single instance. Without venturing even to suggest an explanation of the striking contrast herein presented with the murmur of inadequacy of the aortic valves from ordinary morbid change, I should in future regard it as indicative of failure of the valves from some other cause, most probably mechanical injury.

CASE XXVI.—*Irregular and Suspicious Breathing; Intermittent Pulse, and Great Debility; Polyuria; Death. Granular Degeneration of the Kidneys; Fatty Deposition on the Heart; Simple Hypertrophy of the Left Ventricle.*

John D., aged forty-eight years, labourer, admitted September 25th, 1864. For last three years he had been weak and out of health, and his feet began to swell six weeks prior to admittance. Pulse weak and intermittent, and respiration occasionally accelerated and suspicious in the recumbent posture. Urine passed in large quantity, amounting to more than two quarts in the twenty-four hours, sp. gr. 1.010, and highly albuminous; feet slightly swollen; apex-pulsation somewhat to left of normal position, and both sounds of heart sharp, clear, and free from murmur. At midsternum the first sound was distinctly double; the first element in time being dull, and the second, which immediately succeeds the former, sharp, and "clicking." These characteristics were recognizable only at the end of expiration, when the chest-wall had entirely subsided. To have citrate of iron and quinine (gr. v.) ter die.

The patient gradually became listless, and then somnolent; the stomach became irritable; the appetite ceased, and he quickly emaciated.

For the last fortnight of his life there was intercurrent diarrhoea, and during that time the breath was cold. Œdema had quite disappeared. Respiration was slow and stertorous. He died comatose, November 17th, and on the following day the body was examined. The heart weighed fourteen and a-half ounces; it presented some superficial fat at the base. Right ventricle very thin, but not dilated, and exhibiting in section a layer of fat on its external surface; some decolorized clot in right chambers. Left ventricle thickened, but not dilated; its walls measur-

ing half an inch at base and central portion. Left auricle dilated; valves all strictly normal; muscular structure healthy. Ascending aorta dilated sufficiently to admit three fingers on edge; dilatation commencing above the valves, which were competent. The lining membrane of the vessel was of a cherry-red tint not removable by washing. Kidneys reduced in size, and nodulated on the surface; the right weighed three and a-quarter, and the left three and a-half ounces. The nodules on the surface of the kidneys were large, but slightly elevated, and of much lighter colour than the intermediate surface. In section they were light yellow, as was likewise the greater portion of the cortex. The latter was increased in thickness, and passed in between the pyramids, which were dwarfed. Under the microscope, and with a power of 222 diameters, the Malpighian bodies were seen nearly filled with amorphous matter, which obscured the vascular glomeruli, and yielded negative results when treated with iodine and sulphuric acid.

The primary disease in the foregoing case was renal, which was followed by left ventricular hypertrophy, and, subsequently, by fatty deposition on the heart, and dilatation of the aorta. Simple hypertrophy of the left ventricle, the first change in the pathogenetic series consecutive to granular disease of the kidneys, would have been converted in this case, under the influence of greatly impaired nutrition, into dilatation with thinning of the walls, had the fat been deposited amongst or within the muscular fibres, in place of upon the surface of the heart; because the walls, then weakened in contractile and resisting power, would have yielded under the pressure of the blood from within. I think that such is the history of simple hypertrophy in most instances, as already stated; namely, that it constitutes the first of a series of pathological changes in the heart, the last member of which is dilatation, which accompanies or supersedes hypertrophy according to the state of general or special nutrition.

Under the same circumstances, namely, if tissue-degeneration of the heart had taken place instead of fatty deposition, the aorta would have been dilated at its orifice, and valvular inadequacy would have been the result. Dilatation of the first portion of the aorta, including the orifice or not, constitutes, in my

opinion, the essential condition for the occurrence of the peculiar rhythmical irregularity of breathing known as the "ascending and descending respiration" of Cheyne. This connexion was well exemplified in the case just narrated. Finally, the dependence of reduplication of the first sound of the heart upon *simple* hypertrophy of the left ventricle is illustrated in this case.

CASE XXVII.—*Albuminuria and Anasarca ; Orthopnœa ; Heaving Impulse of the Heart, and Reduplication of the First Sound, with subsequent Development of Mitral Systolic Murmur ; Uræmic Convulsions and Coma ; Death. Cirrhosis of the Kidneys ; Simple Hypertrophy of the Left Ventricle ; Recent Endocarditis of the Mitral Valve ; Contraction of the Aorta.*

Marianne C., aged nineteen years, a domestic servant, admitted October 2nd, 1869. When a child she had severe pain in the region of the heart. Menstruation has been irregular during the last twelve months, and for the last two months it has been entirely suspended. Nine months ago she began to suffer from shortness of breath and palpitation, and three months since she was forced by ill health to relinquish her employment. For the last two months she has not been able to lie down, owing to the feeling of suffocation induced by the recumbent posture. A week since she spat a little blood, and about the same time her feet began to swell. When admitted she was anæmic ; feet slightly puffed ; there was orthopnœa, and great respiratory distress on assuming the recumbent posture even for a moment. Pain shooting down the left arm. Respiration in sitting posture 48 ; pulse 108, weak, but regular ; occasional vertigo ; urine, sp. gr. 1·010, and loaded with albumen. Great increase in extent of precordial dulness ; heaving impulse, and very peculiar action of the heart, consisting of a rolling motion associated with three cardiac sounds, of which the first two were systolic in rhythm, and dull. No murmur. Râles over base of right lung both anteriorly and posteriorly. To have quinine and ether, and an opiate plaster over the heart.

October 10th. Stomach has been very irritable, refusing food and medicine ; dyspnœa much aggravated at night, and threaten-



ing suffocation. A distinct systolic apex-murmur now exists, and only two sounds are audible. To have tincture of digitalis with syrup of the American wild cherry (*Prunus Virginiana*).

25th. Since last report, feet, legs, and genitals have become greatly swollen, but are pale and wax-like. Vertigo; systolic apex-murmur of a soft and blowing character. Amaurosis and coma gradually supervened, the latter very slowly, and were succeeded by a series of epileptiform convulsions, about three in the twenty-four hours, commencing with rapid twitchings of the face, arm, and leg, first of the left side, and then of both sides, and ending with stertorous breathing and profound coma. The pupils were dilated during the stage of clonic spasm, and contracted in that of the consecutive coma. During the intervals between the fits there was partial consciousness or susceptibility to strong sensorial impressions. Deglutition was perfect; alvine and vesical evacuations involuntary.

She died on the evening of the 26th October.

The heart was enlarged, and, with an inch of aorta and pulmonary artery attached, weighed nineteen ounces. There was a large "milk-spot" on right ventricle, and one upon either auricle. The left ventricle was greatly hypertrophied, and constituted an excessive proportion of the volume of the entire organ. It was not dilated; walls about ten lines thick at base and centre, but much thinner at the apex. Mitral valve apparently competent to close the orifice, and structurally sound, save that there was slight thickening of the free edge, and crimson reddening of this, and of the entire ventricular surface of both segments.

Left auricle normal, and mitral orifice of average size. Both left chambers contained a good deal of dark clotted blood. Muscular substance of left ventricle examined microscopically, was found healthy. Aortic valves normal and competent, and aorta healthy, but in calibre somewhat under the normal standard.

Right chambers normal. Right ventricle contained a solid coagulum of a globular figure, and coated on the surface with yellow fibrin; it projected through the tricuspid orifice, and divaricated the valves, but had no connexion in the auricle. Kidneys much reduced in volume, and lobulated on the surface;

the right weighing two and a-half, and the left three ounces. Section showed thickening of the cortex, which was dark and congested; examined microscopically, it exhibited no abnormal appearance, except considerable thickening, with hyaline appearance of the periphery, of the Malpighian bodies. The Malpighian cones were much reduced in size.

As in the case last detailed, the starting point of disease in that just given was manifestly cirrhotic or granular degeneration of the kidneys. From this, or rather from the capillary obstruction consecutive to it, arose simple hypertrophy of the left ventricle.

The occurrence of consecutive endocarditis whilst the patient was under observation, was announced by mitral systolic murmur; and, at the examination of the body, this was proved, by the still recent inflammation of the valves, to have been coeval with the development of murmur. Disease of the kidneys was diagnosed early in the progress of the case, as was likewise simple hypertrophy of the left ventricle; and the convulsions were regarded as uræmic.

Both this and the preceding case afford notable illustrations of the causal connexion between simple hypertrophy of the left ventricle and reduplicated first sound. In both cases this was the only cardiac lesion to account for the phenomenon.

I have stated at length in a previous chapter (p. 164) my view as to the cause of reduplication of the first sound. It consists essentially in a resolution of the sound into its two component elements of apex-shock and valve-tension. If the left ventricle be in volume greatly in excess of the remainder of the heart, without having undergone enfeeblement by proportionate dilatation, or by tissue change, it will strike the chest-wall with vigour and produce a loud but dull sound. Further, the impact of the left ventricle, owing to the energy of its contraction, would conceivably, in such a case, anticipate the tension, though not the closure, of the auriculo-ventricular valves, and so give rise to double first sound. The second element of such a sound is invariably sharp and clicking, such as membranous flaps would yield under strong and sudden tension. The disappearance of the second element of the double sound in the latter of these

cases, simultaneously with the development of mitral regurgitant murmur, tends to confirm the view just stated as to the cause and diagnostic value of reduplicated first sound.

CASE XXVIII.—*Paroxysmal Dyspnœa ; Postdiastolic Basic Murmur ; Sudden Death. Hypertrophy with Dilatation of both Ventricles ; Thrombosis on both Sides of the Heart ; Dilatation of the Aorta, and consequent inadequacy of the Aortic Valves ; Granular Degeneration of the Substance of the Heart ; Cirrhosis of the Liver.*

William H., aged forty-five years, an engineer by profession, in which capacity he served many years on board ship exposed to a temperature varying from 107° to 112° F., but latterly employed on railways, was admitted into hospital, June 26th, 1870. He stated that his habits had been temperate, and that he had been ailing two years prior to date of admittance. Within that period he often had temporary dimness of vision and threatening of syncope, and about the end of the preceding December he vomited some dark blood.

When visited by me on the morning of the 27th, he was suffering from dyspnœa of a paroxysmal character accompanied with puffing, and associated with a feeling of great oppression, referred to the region of the sternum, and expressed by frequent moaning. Feet and hands cold and livid, but not œdematous ; slight engorgement of cervical veins, and slight cough. Pulse 96, feeble but regular, and not visible. A strong and heaving diastolic impulse was perceptible throughout the precordium. Apex-pulsation in nipple line, and in sixth intercostal space, and here a (transmitted) diastolic murmur was audible, but no first sound save a weak impulse element.

At the base the first sound was of a similar character, and a loud and soft postdiastolic murmur existed ; no murmur audible in the neck. To have citrate of iron and quinine in gr. iij doses thrice daily, and chloral hydrate (gr. xv) at night.

On the 29th, his distress continuing unabated, two leeches were applied at midsternum with much relief to the feeling of oppression. At 9 o'clock P.M. he declared he felt much better,

and at 4 o'clock the following morning he was found dead in his bed.

*Post mortem.* Lungs congested; liver in early stage of cirrhosis, and covered with false membrane on the anterior surface to the extent of the palm of the hand. Heart enlarged and elongated, weighing twenty-three and a-half ounces. Aorta dilated, admitting readily the four fingers and thumb, and measuring one and a-half inch in diameter immediately above the valves. It presented patches of atheroma; and, with the exception of the sinuses, where the walls were apparently healthy, the lining membrane throughout was corrugated, and of a pink red hue irremovable by washing. Left ventricle dilated and somewhat thickened, its walls measuring half an inch at the base, but gradually thinning off towards the apex; and here, at a point about one-eighth of an inch in diameter, it was reduced to a thickness of two lines. It contained some dark coagulated blood which passed into the aorta. Mitral valve adequate and perfectly healthy. Aortic valves healthy, but incompetent owing to dilatation of the vessel. The anterior right segment was slightly thickened throughout the *lunula*, and hung on a lower level than the other two. Water poured into the aorta passed rapidly into the ventricle. Right ventricle dilated and thickened, and containing some decolorized clot, which passed from the right auricle through the tricuspid orifice, divaricating the valves, and, by a narrow band, into the pulmonary artery; the valves, walls, and calibre of the latter vessel were normal. Auricles normal as to capacity and thickness, the right containing much decolorized fibrin, which had completely filled the appendix, and passed into the ventricle as already described.

Tissue of heart of a less pinkish tint than natural, and in the "granular" stage of fatty transformation.

The diagnostic value of right basic postdiastolic murmur in regard to the precise state of the aortic valves, is well exemplified in the physical signs exhibited in this case, as explained by the morbid changes discovered after death. The valves were healthy, with the exception of slight thickening of the free margin of one of the segments; they were, therefore, capable of yielding tension-sound (second sound), but incompetent owing to dilatation



of the aorta ; hence the murmur succeeding the second sound. The commencement of ventricular diastole and of aortic reaction is coeval with the closure of the semilunar valves, and denoted by the second sound arising from the tension of these valves, as already explained. Therefore, if the valves be in a healthy condition, quite irrespectively of their competency to close the orifice, a normal second sound will be heard ; but if they be, at the same time, inadequate from any cause not compromising their structural integrity, or only very slightly affecting it, such as deposit upon, or thickening of, their edges, to an extent sufficient to preclude accurate adaptation of the segments ; or if they be inadequate by dilatation of the orifice of the vessel, then a murmur will follow a normal second sound. Such a murmur I designate as of postdiastolic rhythm.

To distinguish between the two causes just indicated as capable of giving origin to a murmur of this rhythm, the paroxysmal dyspnœa, more or less regularly ascending and descending in rhythm, will serve as a positive guide. As stated in my comments on a previous case, I believe this symptom to be pathognomonic of dilated aorta. When, therefore, in association with this symptom, a clear second sound immediately succeeded by a murmur is heard, the diagnosis of valves structurally healthy, but incompetent from dilatation of the aorta, may be confidently made.

The immediate cause of death in the foregoing case I believe to have been thrombosis of the right chambers of the heart, and inadequacy of the tricuspid valve by entanglement of the connecting band of fibrin. The consequence was that the left ventricle, already in a degree incompetent to maintain the arterial circulation, partly owing to its inherent weakness and partly by the excessive dilatation of the aorta, now imperfectly distended with blood from the lungs and inadequately nourished by a failing coronary circulation, ceased to contract, and death was the result of ventricular asystole.

CASE XXIX.—*Anæmia and great Debility; Urine of low sp. gr., and presenting Hyaline Casts; Dropsical Effusion; Death. Cirrhosis of Kidneys; Hypertrophy and Fatty Degeneration of Left Ventricle of Heart, and Dilatation of Right Ventricle.*

Margaret T., aged eighteen years, of respectable birth, was admitted November 29th, 1871. Had never had rheumatism or other serious illness. Her chief complaint was of debility. She was pale and undergrown, and remarkably nervous and choreic in her manner and movements. Had never menstruated.

Under a tonic plan of treatment, including iron and shower-baths, she improved so much that her discharge was contemplated, when, about three months after admittance, the feet and legs began to swell; the stomach now occasionally rejected food, and the pulse became gradually weak and filiform.

The urine was tested and found of a clear, amber colour, acid, sp. gr. 1.010, and free from albumen. The precipitate examined microscopically exhibited epithelium with a few transparent tube-casts. The face soon became swollen, and the breathing much embarrassed, whilst the pulse was hardly perceptible.

Precordial dulness seemed to be extending; no cardiac impulse could be detected, and the sounds of the heart were faint and distant, but free from murmur. The cervical arteries pulsed visibly, and the face and lower limbs became livid.

She died on the 15th March, 1872.

On examination of the body both pleuræ were found quite full of serum, whilst the pericardium contained only about one ounce. The heart was much enlarged, weighing fifteen and a-half ounces; the left ventricle dilated and thickened, but the right ventricle rather attenuated. Examined microscopically, the structure of the ventricles was found to be in the granular stage of fatty degeneration; the transverse striæ had disappeared, and the section was dotted with large oil-drops.

The valves of the heart were all normal, and the foramen ovale was closed. Kidneys lobulated on the surface and cirrhotic, but not reduced in size; the left weighing four, and the right four and a-half ounces. The cortical structure was rather thicker than normal, firm, and light in colour, but the pyramids

were reduced in size and congested. A section of the cortex examined microscopically presented the capsules of the Malpighian bodies greatly thickened, and composed of concentric laminae of fibroid texture, whilst the glomeruli were so much dwarfed as to be scarcely discoverable; some thick puriform fluid was found in the calices. The liver was large and congested; the spleen small; no liquid in the peritoneum.

An enlarged heart, weakened by fatty degeneration, is not unlikely to be mistaken for hydro-pericardium, more especially if associated with hydro-thorax and slight effusion into the pericardium itself; all which conditions existed in this case. Trousseau adverts to the difficulty of diagnosis, even in the absence of the last mentioned condition.

The case was manifestly one of primary renal disease with consecutive hypertrophy of the left ventricle, and, later on, fatty degeneration of the hypertrophied heart.

The consecutive change of fatty transformation of the heart was the sole cause of failure of the heart and pulse, and of congestion of the lungs and extremities. It was likewise a contributory cause of the dropsical effusion.

The absence of albuminuria in connexion with cirrhosis of the kidneys, is worthy of special notice.

*CASE XXX.—Albuminuria; Left Hemiplegia, and Mitral Systolic Murmur; Paroxysmal Dyspnoea; Erysipelas; Uræmic Coma; Death. Cirrhosis of the Kidneys, Liver, and Spleen; Hypertrophy of the Left Ventricle, and General Fatty Degeneration of the Heart; Fibrinous Deposit on the Mitral Valve; Dilation of the Aorta.*

Robert R., aged forty-five years, house-painter; has had a slight attack of painter's colic. Admitted May 4th, 1872. In the course of the preceding summer he suffered from dry retching in the morning and from headache, and shortly afterwards he suddenly lost the use of the left arm and side of the face; but the left leg was affected only in a very slight degree. At the end of six weeks he had so far recovered the use of his arm as to be able to resume his work. In March, 1872, he had a severe

attack of epistaxis; and he noticed, for the first time, that his breathing was affected.

When admitted he was remarkably pallid; face slightly drawn to the right, and on the left side the natural creases were nearly obliterated. The tongue, when protruded, deviated much to the left, and the left arm and leg were weak. Urine albuminous and containing granular tube-casts; œdema of feet; pulse 104, regular; radial arteries rigid and remarkably tortuous, that of the right side making several curves fully an inch in depth. Temporal arteries likewise tortuous and rigid, and in both radial and temporal arteries pulsation was visible. Heart's action weak; apex-pulsation faintly perceptible in nipple-line, and at this point a bellows-murmur of medium intensity accompanied the first sound. At the base the first sound was obscure, and the second sound remarkably sharp and ringing, a character which it preserved throughout the arch of the aorta. Alternations of paroxysmal and suspended breathing; cough and purulent expectoration, and physical evidence of engorgement of right lung. He had several attacks of vomiting during his residence in hospital, and on the 25th May erysipelas of the face, which, under the persistent application of an unguent containing 3j of sulphate of iron to 3j of simple ointment, disappeared in the course of two days.

1st June he became comatose, and on following day he died.

*Post mortem*, June 3rd. A small quantity of turbid serum was found in the cavity of the peritoneum. The liver was hard and furrowed, and the spleen was in a similar condition, but rather small, the fibrous element being greatly in excess; kidneys contracted and cirrhotic. The lungs were congested, but there was no serum in the cavities of the pleuræ or pericardium. The heart was much enlarged, somewhat flabby, and globular in figure; it weighed twenty-three and a-half ounces. The aorta was dilated and patchy with atheroma; but the lining membrane was natural in colour, and the sigmoid valves healthy and competent. The right ventricle contained a flake of yellow fibrin, which extended in form of a cylinder into the pulmonary artery, half filling the vessel; it was normal as to size and thickness of walls, but its component muscular tissue was in the granu-



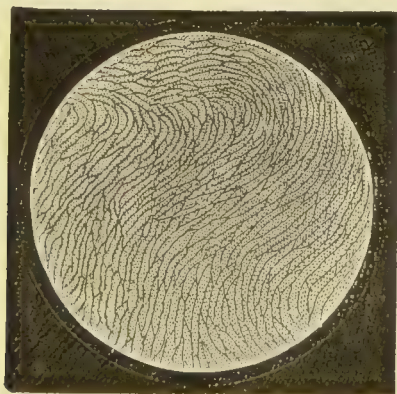
lar stage of fatty degeneration; presenting the appearance of lines of translucent dots, and only in one spot in the field, a trace of transverse striation. There was no free fat. (See engraving, Fig. XXXI.) The right auricle was likewise filled with decolorized fibrin, which descended into the inferior cava. Left ventricle somewhat dilated, and its walls more than an inch thick in the central portion. The septum projected to the right side. Mitral valve somewhat thick and opaque, but pliant, and competent to close the orifice; some flakes of fibrin were entangled in its edge. The muscular structure of the left ventricle was likewise in a state of fatty degeneration, only somewhat less advanced than that of the right; the transverse striæ being more extensively and more distinctly visible.

Renal disease was likewise the primary affection in this case; and hypertrophy of the left ventricle, fatty transformation of the substance of the heart, and aortic and general arterial athetoma, with dilatation of the aorta, were subsequent changes.

The cause of hemiplegia and of mitral systolic murmur was to be found in thrombosis of the left ventricle, entanglement of a detached flake of fibrin in the margin of the mitral valve, and arterial embolism of the right motor nerve-centre.

The absence of aphasia, in connexion with *left* hemiplegia and the coexistence of rhythmical irregularity of respiration with dilatation of the aorta, without vascular injection of its lining membrane, should be noticed.

FIG. XXXI.



Granular degeneration of muscular substance of right ventricle,  $\times 120$ . Case of Robert R. This engraving should be examined with a pocket-lens.

CASE XXXI.—*Great Debility and Pallor; Œdema; Visible Pulsation at the wrist; Slight Displacement of the Apex of the Heart to the left; Systolic Apex and Basic Murmur; Systolic Murmur at Ensiform Cartilage; Loud Arterial Murmur; Sudden Death after convulsive seizure. Serous Effusion into Pleuræ and Pericardium; Fatty Deposition upon, and Granular Degeneration of, Right Ventricle of the Heart; Hypertrophy and Dilatation of Left Ventricle; Flap of Fibrin attached to Ventricular Surface of Mitral Valve and floating into Orifice of Aorta; Dilatation of Tricuspid Orifice.*

Mary D., aged forty-eight years; admitted into hospital June 20th, 1872. Had been short of breath, and generally out of health for some time. Examined on the morning of the 21st, the following was found to be her condition. She was remarkably pale but in no degree cyanosed, and extremely weak; feet much swollen; radial pulse 84, very weak, visible, but regular; urine loaded with lithates, sp. gr. 1.025, and free from albumen. The apex of the heart pulsated slightly to the left of the normal position very feebly, and at this point a loud bellows-murmur accompanied the first sound. At the base a systolic murmur, but more harsh in quality, was likewise audible, and was transmitted thence through the arch of the aorta with great intensity, and also distinctly into the carotids; second sound weak, but normal in both these situations. At the ensiform cartilage the sounds were remarkably sharp and flapping, and here a soft systolic murmur existed.

The chest was resonant in front, and respiration was normal; posteriorly the base was dull on both sides, and the respiratory sounds were feeble.

On the evening of that day (21st) she died suddenly after a convulsive seizure, and on the following morning the body was examined.

There was a good deal of serum in both pleural cavities, and some cretified tubercle in the apex of the left lung, but no adhesion of the pleuræ. There was nearly a pint of serum in the sac of the pericardium, and a considerable deposit of fat on the

surface of the heart, which was slightly in excess of the normal size. The right auricle was dilated; the right ventricle greatly dilated, its walls flabby, and not more than two lines thick in any portion of their extent. In section the outer third was seen to be composed of fat; and the remainder, microscopically examined, was found to be in the granular stage of fatty transformation. The tricuspid orifice was greatly dilated, readily admitting the four fingers and thumb to pass through, and the tricuspid valve somewhat thickened, but pliant and expanded. Both right chambers contained some decolorized fibrin, which extended a short distance into the pulmonary artery. The left auricle was dilated and somewhat thickened. The walls of the left ventricle were greatly thickened, but healthy-looking and firm; its cavity was slightly dilated at the apex, but elsewhere it was of normal dimensions. The anterior segment of the mitral valve was much thickened throughout, irregular at the edge, and incompetent; and on its ventricular surface, about three lines from the free margin, there was attached a tongue-shaped flap of fibrin, about a quarter of an inch in length, and organically united to the valve. When the ventricle was filled with water this flap was floated into the orifice of the aorta, and, during ventricular systole, must have produced by its vibration an eddy in the efflux current, and a systolic basic murmur propagated into the great arteries. The aorta was perfectly healthy; lungs congested posteriorly; other viscera normal.

Mitral valve-disease was in this case the primary affection; giving rise to hypertrophy and dilatation of the left ventricle and auricle, pulmonary congestion, and dilatation of the right ventricle and tricuspid orifice. Fatty deposition upon, and granular degeneration of, the right ventricle, were subsequent changes, and, no doubt, still further impaired its functional capacity, and were the immediate cause of dropsy. The most interesting feature in the case, diagnostically, is that of aortic and carotid murmur due to fibrinous deposit upon the *mitral valve*.

Doctor Flint has recently directed special attention to this phenomenon. I think, however, he over-estimates its frequency. I have no record of a second example of the kind, proved to be such by dissection.

The origin of the epileptiform seizure, which was the immediate cause of death, would probably have been found to be extensive capillary embolism of the brain, had the contents of the cranium been examined, which, I regret, was not the case.

In regard to the relative frequency of hypertrophy from the several causes mentioned, and the average duration of life in each of these categories, the following summary of the preceding fourteen cases may possess some value :

<i>Cause.</i>	<i>No. of Cases.</i>				<i>Average Duration.</i>	<i>Result.</i>
Renal disease ...	...	7	...	10 months (nearly)	} Death.	
Dilatation of aorta ...	...	2	...	24 „		
Valvular disease ...	...	3	...	8 „		
Rupture of aortic valve ...	...	1	...	3 „		
Rupture of mitral valve ...	...	1	...	3 „		

It will be observed that the renal form of the malady has furnished quite one-half of the total number of cases given.

It should be noted, however, that this is not due to an absolute preponderance of hypertrophy of renal origin ; but rather to the fact, that with this form of the affection, owing to the attendant dropsy, patients are more alarmed about their health and less able to shift for themselves, than they are with hypertrophy from any other cause. They consequently seek hospital relief in greater numbers, and yield a larger comparative mortality.

The duration of life was greatest in those cases in which hypertrophy arose from dilatation of the aorta, and least in those in which it was caused by ruptured valves. This difference was, no doubt, due to the slow progress of atheroma and dilatation of the aorta, during which the left ventricle had time to acquire a proportionate increase of volume and of propulsive power ; whereas, in the case of rupture of the valves, the ventricles were surprised, so to speak, and incapable of carrying on the circulation under the increased difficulty suddenly opposed to them.

*Dilatation* of the chambers of the heart, or "aneurism," as it was designated by Lancisi, was divided by Corvisart into "active" and "passive;" the former corresponding to the "dilatation with hypertrophy" of modern writers, and the latter to "dilatation with thinning" of our nomenclature.



Morgagni gave many examples of both forms. To these Bertin added two others, namely, "simple dilatation," in which the walls are normal as to thickness; and "mixed dilatation," in which portions of the walls are thinned, others thickened, and others again are normal. Bertin held that dilatation of any of the chambers of the heart necessarily implied an obstacle to the circulation in front of it.\* He thus ignored that form of dilatation, now so common, in which, without obstruction to the circulation, simple debility and softening of the walls of the heart from anæmia or from retrograde changes of tissue, becomes a cause of yielding and dilatation. He commits the further error of assuming that vascular engorgement, passive hæmorrhage, and anasarca, when associated with dilatation, are due to the assumed vascular obstruction which he believes to be a necessary antecedent condition, rather than to dilatation. These consequences ensue, however, only when the heart fails to overcome the obstacle in front, normal or abnormal, and becomes dilated.

The classification adopted by Hope is unexceptionable save as to completeness.† He divides dilatation into three forms, namely, "simple dilatation," in which the chamber is dilated whilst the walls are of normal thickness; "dilatation with hypertrophy," the cavity being dilated and the walls thickened; and "dilatation with attenuation," the cavity being dilated and the walls thinned. To these I think the "mixed" form of Bertin should be added; because examples are of not rare occurrence in which one or more portions of the walls, especially of the left ventricle, are normal or actually thickened; whilst at other points, usually near the apex, the wall may be reduced to the utmost tenuity.

Dilatation with comparative rarity engages all the cavities of the heart; and never, save where it is the result of a general or constitutional cause, such as typhoid or fatty softening, anæmia, etc. Hope and Walshe maintain, on the contrary, that dilatation is more frequently general than partial.

But if the frequency with which dilatation, in some degree, of one or more of the cavities is met with, be considered, I am

\* *Traité des Maladies du Cœur*, p. 382.

† *Opus citat.*, p. 294.

convinced the partial or local will be admitted to preponderate numerically over the general form of the affection. If, for example, cases of chronic bronchitis with periodic exacerbations, in which the right ventricle is more or less dilated, and those of primary emphysema of the lungs leading to a similar result, be admitted into the calculation, as they must be; and if the numerous examples of dilatation, in some degree, of the left ventricle and left auricle, in connexion with valvular disease at the mitral or aortic orifice, and accompanied or not by hypertrophy, be added to the preceding list, a moment's reflection will suffice to show that the sum of such cases treated by any hospital physician greatly exceeds the total of those of general dilatation which have come under his personal notice.

Of much greater value than the pathological classification just given, is the clinical division into "primary" and "consecutive" dilatation.

The former comprises cases arising from :

- (a) Anæmia.
- (b) Over-exertion and malnutrition combined.
- (c) Excessive innervation.
- (d) Inflammatory, typhoid, or fatty softening.
- (e) Excessive use of tobacco.
- (f) Onanism.

In the "consecutive" form dilatation may result from :

- (a) Obstruction or regurgitation (or both) at the orifice of the aorta.
- (b) Obstruction or regurgitation at the mitral orifice.
- (c) Atheroma of the aorta.
- (d) Obstruction or regurgitation (or both) at the orifice of the pulmonary artery (very rare).
- (e) Renal disease.
- (f) Obstruction to the pulmonary circulation.

With regard to the mutual relationship of hypertrophy and dilatation, it may be said generally, that when both these states coexist, hypertrophy has been the antecedent, dilatation the consecutive change. Hypertrophy implies increased action of the chamber which has become hypertrophied, and proportionate activity of nutrition; but the causes of hypertrophy, in the

consecutive form at least, are always progressive. Hence, hypertrophy, which is but the measure of resistance to retrograde pressure, is likewise in this form of the affection progressive, so long as the processes of nutritive change in the heart may be quickened. A period, however, ultimately arrives when, although resistance in front continues to increase, the compensating process of hypertrophy of the corresponding chamber of the heart ceases, because interstitial nutrition of its walls fails, either from impairment of the conducting properties of the coronary arteries by atheromatous change of their coats, or from simple starvation of the tissue of a thickened heart by its distance from the nutrient centres, namely, the coronary capillaries. Once this derangement of balance has occurred, and its occurrence is inevitable sooner or later, what Professor Flint appropriately designates the "vital process" of hypertrophy is replaced by the "mechanical process" of dilatation.\* Nay more; the form which in any given case a heart undergoing this series of changes actually presents, essentially depends upon the stage of the process at which it is examined, and the stage of thickening at which nutrition of the heart fails. Thus, examined in the early stage, before nutrition had begun to fail, a state of simple hypertrophy, *i.e.*, thickening of the walls, without alteration in the size of the chamber would be found. At a later period thickening may be found still greater, but as yet no change in the capacity of the chamber, a condition differing only in degree from the preceding; or the process of hypertrophy may have ceased owing to early failure of nutrition, and the condition known as moderate hypertrophy with dilatation, or dilatation with hypertrophy, according as one or the other condition preponderated, would be found to exist. In a case where nutrition had progressed to an advanced period, *pari passu* with mechanical resistance to the circulation, and then failed, the heart would be found greatly hypertrophied, with a moderate degree of dilatation; or, at a still later period, disproportionately dilated. The former of these conditions would constitute "hypertrophy with dilatation," and the latter "dilatation with hypertrophy." Simple dilatation and dilatation with thin-

\* *Diseases of the Heart*, second edition, 1870, p. 79, *et sequent.*

ning of the walls are usually primary, although, theoretically, a state of extreme dilatation with or without thinning, may represent the last member of a series of retrogressive changes in a heart previously in the highest degree hypertrophied. Such a heart would be greatly enlarged; but on this subject I have no positive information to offer.

I cannot accept the pathological dogma of Rokitansky,\* that dilatation is the natural result of valvular regurgitation; whereas hypertrophy follows obstruction. It would be more correct to say that hypertrophy is a *primary*, and dilatation a *secondary*, result, equally of obstruction and regurgitation. No doubt, partial dilatation of the left ventricle at the apex may coexist with hypertrophy of the remainder of its walls in permanent patency of the aortic valve, owing to the long continued pressure which it has had to sustain from the reflux column of blood during diastole. But even here, the dilatation is consecutive to hypertrophy. In what has preceded, it is, of course, implied that hypertrophy can in no instance be secondary to dilatation.

*General* dilatation of the heart is always of constitutional origin, and the result of one or other of the causes of "primary" dilatation already mentioned, although these causes may be secondary in operation.† Thus, it may result from anæmia, and be "primary," in one case; and from the same cause, and yet be "secondary" to valvular lesion and simple hypertrophy in another. Again, it may be due to "primary" fatty degeneration, or to fatty degeneration "consecutive" to hypertrophy from valvular lesion or from chronic renal disease. The distinction is important; because, as I will hereafter show, the latter or secondary form of general dilatation, is by much

\* *Pathological Anatomy*, vol. iv., p. 162.

† Doctor Stokes many years ago foreshadowed these views, in the following paragraph, which came under my notice only when the preceding passage had been written. I need not say how much pleasure it afforded me to find my views sustained, because anticipated, by the great authority of my illustrious teacher. "It is still to be determined whether the cavities of the heart are liable to change from mechanical causes alone; whether dilatation, for example, is a purely mechanical result of obstruction to the exit of the blood, or whether for its production in valvular disease there is required not only obstruction, but a weakened condition of the heart." (*The Diseases of the Heart and Aorta*, 1854, p. 255.)



the more serious prognostically, and the more rapidly fatal of the two.

Beau, indeed, maintains that general dilatation with hypertrophy of the heart is the legitimate pathological offspring of pericarditis issuing in universal adhesion of the pericardium; the false membranes, by their contraction, exercising a general dilating force upon the heart.\* In reference to this theory, regarding which Beau declares that "he cannot adopt it in an absolute manner," it may be remarked, that where the pericardium is not extensively adherent to the chest-wall, there is no sufficient *point d'appui* whence the false membranes can exercise a general excentric traction upon the heart. All such cases I believe to be examples of previous hypertrophy, from whatever cause, with subsequent pericarditis and inflammatory softening and dilatation of the heart.

The *symptoms* and *signs* of general dilatation of the heart will be found to vary within certain limits, according to the stage at which it is examined. Thus, at an early period of its history, yet when the characteristic features are sufficiently pronounced, the patient will exhibit languor of mind and body; the extremities, ordinarily of a slightly livid tint, will be readily chilled and benumbed; the pulse, small and regular, may intermit at long intervals; respiration is normally shallow, and readily quickened by exertion, and the patient is easily put out of breath by physical effort, or mental excitement. There is a constant hemming or teasing cough, accompanied by scanty serous expectoration and a proclivity to catching cold, which constitutes the patient's principal infirmity. On these occasions there is great embarrassment of breathing, which may amount to orthopnoea; the pulse is quick, feeble, and it may be irregular; the conjunctivæ injected with dark blood, and the features generally livid and somewhat puffed.

In the ordinary condition of the patient secretion and excretion are performed with regularity, although the liver is somewhat engorged, and the patient may be troubled with hæmorrhoids. The appetite, however, is indifferent, and digestion is slow; and from time to time, as shown by foul tongue

\* *Archives Générales de Médecine*, vol. x., April, 1836.

eructation and anorexia, the stomach suffers derangement of function, either as a consequence of some slight indiscretion in regard to food, or in damp and foggy weather without any such provocation. On such occasions the urine precipitates a copious sediment of lithates. The extent of precordial dulness is horizontally increased, if not masked by emphysematous lungs. The impulse of the heart is feeble, diffused, and occasionally intermittent; and a distinct point of apex-pulsation can rarely be detected. The precordial region, viewed in profile, will generally exhibit indistinct fluctuation in one or more intercostal spaces. The sounds of the heart are sharp and distinct, the first closely resembling the second in brevity and pitch, but varying as to clearness and intensity according to the state of the cardiac walls; it is feeble and low toned, though well defined, when the walls are fatty or otherwise softened, but clear and ringing when tissue-change does not exist. The pulsation of the heart is quickened by even the slightest movement of the body, and on such occasions it is attended with throbbing of the carotid arteries, and visible fluctuation of the external jugular veins.

In a few such cases, as I have elsewhere shown,\* the first sound at the apex is accompanied or succeeded by a soft bellows-murmur, due, as I believe, to inadequacy without disease of the mitral valve. If, at the acme of systole, a portion of the walls of the ventricle to which one or more of the papillary muscles are attached, yielding under the centrifugal blood-pressure owing to its softened or attenuated condition, is carried outwards at an angle from the axis of the ventricle, it is readily conceivable that one of the flaps of the mitral valve may be thereby lifted off the opening through the medium of the tendinous chords, and so give rise to mitral reflux and murmur. Under treatment, such murmur may cease coincidently with the occurrence of general improvement in the condition of the patient (see Cases 36 and 37).

Doctor Walshe would regard regurgitation in such cases as due to general dilatation of the left chambers and mitral orifice,

\* *British Medical Journal*, December 18th, 1867.

and consequent inadequacy of the valves ; but manifestly, if the auriculo-ventricular ring were dilated, it would, owing to its tendinous structure, be incapable of recovering its former dimensions under tonic treatment, so as to adapt itself to the inadequate valve and prevent further reflux. Yet, if we will not admit that the valve itself may have undergone expansion to the requisite extent to fill the opening, and that this process may have been, in a series of cases, coincident with treatment rather than consequent upon it (for the treatment that has been successful in removing the murmur could have no effect in expanding the valve), an assumption which I am by no means prepared to grant, we shall be forced to believe that such changes of expansion and contraction of the tendinous ring have actually taken place. I repeat that, in my opinion, such cases are explained by the existence of localized softening, or extreme attenuation of the wall of the left ventricle.

When general dilatation has attained its maximum, respiration is shallow, laboured, and protracted, and usually accompanied by a loud wheeze ; there is complete orthopnoea, and impending suffocation when the patient assumes the recumbent posture even for a moment. The features, especially the lips, tip of the nose, and lobes of the ears, are livid, the conjunctivæ congested and somewhat jaundiced, and the eyelids and face generally bloated ; surface and breath cold ; the feet cold, puffed, and congested. The pulse is slow, feeble, intermittent, and irregular. Precordial dulness extended horizontally, but difficult to be defined with accuracy, because the anterior borders of the lung, which are usually emphysematous, overlie the precordium to some extent. The heart pulsates at the ensiform cartilage ; its action is feeble, flapping, irregular, and barely perceptible to the hand ; and if it be not entirely overlain by the edges of the lungs, a fluctuating movement of its anterior wall is perceptible in one or more of the intercostal spaces, at the end of expiration. The sounds are faint, but sharply defined and high pitched, and if the walls be thinned but not softened, the first sound so closely resembles the second in quality as to be distinguishable from it only by its rhythm, as determined by the cardiac impulse or carotid pulsation. Fine crepitant râles are audible

over the bases of the lungs, which are somewhat dull, and the chest generally is either non-vibratile or very feebly so. There is constant teasing cough, accompanied by thin mucous and frothy expectoration frequently tinged with blood.

Anasarca, and serous effusion into the cavities of the chest and abdomen quickly supervene, the latter being usually the immediate cause of death. In such case the mode of death is by coma, resulting from the combined operation of retained renal and pulmonary excreta. Another mode of death is, however, not unfrequently exemplified in this affection; namely, that by thrombosis of the right side of the heart and pulmonary artery.

The conditions upon which this accident, for as such it may be regarded, depends, are partial stasis of the blood by failure of contraction in the right chambers of the heart, and by impairment of respiratory and nutritive attraction arising from feeble respiration and arrested tissue-change.

The symptoms which indicate its occurrence are, a sense of precordial oppression, gradually increasing to a point of great intensity, and attended with tumultuous but feeble action of the heart; great dyspnoea and craving for air; restlessness and sighing; the patient starts from his slumber with great alarm, and is entirely deprived of refreshing sleep; the pulse is quick, weak, irregular, and fluttering, or all but entirely suppressed at the wrist; the surface is cold, damp, and livid; respiration is rapid, full, and laboured, but manifestly ineffectual, although air freely enters the lungs, and loud rhonchi or wheezing sounds are heard all over the chest. The carotid arteries pulsate with great violence, and the jugular veins are turgid and fluctuating. The most characteristic symptom of this condition is, however, the *besoin de respirer*. The arms are thrown out and extended; the covering is flung off the chest; the patient begs that fresh air may be admitted, and declares he is being suffocated, although the chest moves freely and extensively, and there is abundant circulation of air in the chamber, and, as judged by auscultatory evidence, in the lungs also. In this condition death usually occurs quite suddenly on the patient's making the slightest exertion, such as that of sitting up; it is the immediate consequence of the impaction of a mass of fibrin in the tricuspid



orifice or the pulmonary artery, by which the circulation in the lungs is mechanically arrested, the systemic arteries emptied, and the veins gorged with blood. General dilatation of the heart is least promising where the organ has undergone change of structure; it is of unfavourable augury in proportion to the extent and degree of tissue change which the heart has suffered. That of fatty degeneration is the most serious, because the least amenable to treatment; it is likewise the most liable to terminate in sudden death by thrombosis or rupture.

That form of the affection which is secondary to hypertrophy from valvular lesion, or from renal disease, is likewise proportionately more serious than that of primary origin, because of the retrogressive tissue-change which it implies, and the formidable mechanical resistance which, in either case, the heart, thus weakened by tissue-decay and by thinning of its walls, has to overcome in carrying on the circulation.

General dilatation from anæmia, or from excessive labour and inadequate nutrition, is, on the other hand, the most promising form of the affection, and susceptible not only of improvement, but of cure under appropriate and persistent treatment. Dilatation from "typhoid softening" is usually met with in typhus fever, or consecutive to it, and is likewise amenable to treatment.

I have met with only a few, certainly not half a dozen, examples of it in the typical form described by Dr. Stokes, although it must have been not rare at the time he wrote.

*Treatment.*—In the anæmic form, iron is, *par excellence*, the remedy. It should be given in such form as the stomach can best tolerate, and its efficacy will be greatly enhanced if it be combined with strychnia and quinine, as in the syrup of the triple phosphate, of which a teaspoonful may be given in twice that quantity of cold water thrice daily. Where dilatation is the result of excessive physical toil, worry, and malnutrition, as so frequently exemplified in poor women of weak frame and nervous temperament, digitalis and iron combined act as a specific (*vide* Case 37, Mrs. C.) I usually prescribe ℥v-x of the tincture of digitalis with ℥x of the tincture of perchloride of iron, and ℥v of chloric ether, in an ounce of water every third hour.

Nutritious diet, including a large proportion of animal food and a liberal allowance of wine, are at the same time ordered. The bowels, which are usually constipated, but may be over-relaxed, should be attended to. In the former contingency, grs. v of the compound aloetic pill, with a-quarter gr. of extract of nux vomica, may be given every night; and in the latter, ℞ of dilute sulphuric acid, or of the liquor of pernitrate of iron, in an ounce of water, thrice or oftener daily.

In that form of dilatation which results from softening, whether inflammatory, typhoid, or fatty, I rely chiefly upon quinia and strychnia combined; ℞ of the liquor strychniæ of the Pharmacopœia, with grs. iss to ij of quinia, in an ounce of water, acidulated with dilute sulphuric acid and sweetened to the taste, should be given every third or fourth hour. Nutritious diet and an ample allowance of wine should be provided; moderate exercise in the open air should be taken daily; but the ascent of steep, and indeed all causes of excitement of the circulation, whether mental or bodily, and of fatigue, should be studiously avoided.

Doctor Stokes was the first to point out the great benefit to be obtained from the use of mercury in the treatment of fatty and dilated heart, with engorgement of that organ, the liver and lungs, paroxysmal dyspnœa, and anasarca. There seems some reason to conclude that these attacks are associated with latent gout. Dr. Stokes gives, in illustration, the case of the late eminent surgeon Abraham Colles, who ultimately died of this affection. Writing of Mr. Colles, he says: "He continued to suffer from time to time, from paroxysms of dyspnœa, which were generally preceded by diminution in the secretion of the kidneys. During these attacks, which generally lasted for several days, the irregularity of the heart and the precordial distress increased until orthopnœa was established. The kidneys acted scantily, and no copious sediment appeared in the urine. On each attack, the tumefaction of the liver increased with great rapidity; but this condition as rapidly subsided with the improvement in the symptoms. No relief was ever obtained until a free action of the kidneys was established; but it was found that this could only be effected by the use of mercury, followed by diuretics.

On several occasions the diuretic treatment, not preceded by mercury, was tried; but it always failed, so that the number of times in which a distinct course of mercury was employed, was very great. To this remedy, in a great degree, must the prolongation of Mr. Colles' life be attributed, for, on various occasions, the symptoms had gone so far as to cause complete orthopnoea, with unusual anasarca, and alarming pulmonary congestion."\*

I have no personal experience of the use of mercury in cases such as that described by Dr. Stokes; but were an example of the kind placed under my care, I should have no hesitation in adopting the plan of treatment so successfully followed by him.

I have repeatedly observed temporary irregularity in the action of a dilated heart to be directly due to derangement of stomach, as manifested by flatulence, acid eructations, and foul tongue. Dr. Stokes has likewise noticed this, and truly remarked that the cardiac irregularity may be promptly corrected by measures directed to the gastric derangement. I think cases of this kind are also associated with the gouty diathesis, and under the use of acetate of potash and tinct. of colchicum, grs. xxx of the former with ℞ of the latter, given every third hour in an ounce of orange-flower water, and preceded by a saline purgative, I have found the regularity of the heart's action restored in the course of twenty-four to forty-eight hours.

For the relief of precordial oppression arising from excessive cardiac distention, the application of two or three leeches over the base of the heart is the measure most likely to be efficacious for the time. I have repeatedly seen patients who had been entirely deprived of sleep for several nights in succession by this feeling of oppression and impending suffocation, enjoy tranquil and refreshing sleep for several hours after the application of two leeches to the precordium.

In the treatment of the alarming condition due to thrombosis of the heart, in addition to rest, warm applications to the feet, and wine or spirits in small and oft repeated doses, I would make trial of the plan recommended by Dr. B. W. Richardson,†

\* *The Diseases of the Heart and the Aorta*, p. 297.

† See *Medical Press and Circular*, of November 20th, 1872.

although I have had no actual experience of it; namely, that of giving ℥x of the spirit of ammonia in an ounce of water every hour, and grs. v of the iodide of potassium, every alternate hour.

The treatment of dilatation associated with persistent palpitation of the heart and enlargement of the thyroid body, with or without prominence of the eye-balls, constituting the morbid condition known as Graves' disease, and affording a typical example of dilatation from excessive innervation, should be conducted mainly with reference to the general condition of the patient. That condition is preeminently one of debility, and most frequently of anæmia likewise. Hence the indication for alcoholic stimulants in moderate quantity, iron and digitalis, and regimen of a bracing character. I usually allow four to six ounces of sherry daily, and prescribe tincture of the perchloride of iron with tincture of digitalis, of each, ℥x thrice daily, in an ounce of water. For the relief of intercurrent precordial distress I direct two leeches to the precordium, and afterwards an opiate plaster. In cases of extreme nervous susceptibility I have given half drachm doses of bromide of potassium. In all cases I recommend cold shower baths and moderate exercise out of doors; and, when it can be afforded, the use of chalybeate waters for some months in a bracing climate, such as Harrogate.

Dilatation resulting from the excessive use of tobacco is characterized, most frequently, by slight intermittence of the pulse and a tendency to syncope; likewise by a fugacious systolic murmur at the apex of the heart, the action of which is usually quick, but variable as to rate independently of physical exertion. The patient is pale, fidgetty, and generally "nervous," and he gives, when questioned, the history of excessive indulgence in tobacco-smoking. It is, in many cases, idle to recommend a total discontinuance of the practice of smoking. The wiser course is to urge upon the patient gradually increased restriction in this respect, and the use of the mildest "smoking-mixture" obtainable. In addition, cold shower baths should be recommended, and, medicinally, strychnia and iron. For example, ℥v of the liquor strychniæ, with gr.iss of the granulated sulphate of iron in an ounce of water thrice daily.

Onanism, and the cardiac debility and dilatation which result



from it, and which are characterized, as in the preceding example, by nervous excitability and palpitation, and not unfrequently by systolic apex-murmur without valvular disease, are best treated by absolute prohibition of the vicious habit to which they are due; and this should be enforced, if necessary, by vesication of the genitals, or by some equivalent expedient. Bromide of potassium in full doses, 3ss to 3j, is, *par excellence*, the medicinal agent most to be relied upon. It should be given in the above mentioned quantity, thrice daily, for several weeks consecutively, with occasional intervals of one or two days, for the purpose of avoiding the general depression and nausea likely to arise from its long continued use.

Dilatation arising from any of the preceding causes, though general, may affect the two sides of the heart or the individual chambers unequally. Thus, fatty softening is essentially ventricular, rarely engaging the auricles in a great degree, and usually affecting the ventricles very unequally. Typhoid softening likewise most frequently affects the left ventricle in a greater degree than the right, and the consecutive dilatation exhibits in either case a corresponding extent and rate of progress.

Dilatation from nervous debility and habitual excitement, as exemplified in Graves' disease, always exhibits a preponderance in the left, as compared with the right ventricle.

*Consecutive dilatation*, or that arising from obstruction or from a local, as distinguished from a general, cause, is nearly always secondary to hypertrophy, as previously shown. It occasionally happens, however, that the process of atheromatous change in the orifices and valves of the heart, or in the aorta, commences simultaneously and proceeds concurrently with that of fatty degeneration of the substance of the heart. In such a contingency dilatation of the chamber undergoing retrogressive change of its walls, provided the obstruction lay directly in front of it, would be "primary." But that this is of exceedingly rare occurrence may be inferred from the rarity of dilatation without thickening of the walls, in connexion with atheroma of the valves or of the aorta.

Consecutive dilatation, as depending on a local cause, is itself most frequently local in the first instance. It may, however,

affect both sides of the heart at the same time ; but in such a case two concurrent causes, one affecting each side, must be in operation : a contingency exceedingly rare. Not so, however, that of retrogressive dilatation when the disease commences on the left side. In this case, owing to obstruction to the pulmonary circulation, the right side of the heart would be affected successively with hypertrophy and dilatation.

When dilatation of the right ventricle is the result of primary obstruction in the lungs, as when it follows emphysema with oft repeated intercurrent bronchitis, the retrogressive process is arrested in the systemic veins, and never, in my opinion, extends beyond the capillaries to the left side of the heart. I have not met with a single example which would constitute an exception to this statement. Pulmonary obstruction, to an extent and degree adequate to the production of general venous engorgement, when not rapidly fatal, is always intercurrent and temporary.

The systemic veins being remote from the seat of obstruction, and not having suffered permanent impairment of tonicity by temporary distention, readily recover their normal calibre when free circulation through the right side of the heart and the lungs is restored ; and the left heart, thus relieved from backward pressure through the capillaries, will not have undergone from the temporary obstruction thence arising, any alteration either in regard to the thickness of its walls, or the capacity of its chambers. Not so, however, the right side of the heart, which, owing to its proximity to the seat of obstruction in the lungs, and the feebleness of its walls, is found, after repeated distention, to have become permanently dilated.

The rule may be therefore regarded as general, that whilst obstruction on the left side of the heart is competent to produce hypertrophy and dilatation of the right side ; primary stasis on the right side is not reciprocally productive of hypertrophy and dilatation on the left side.

*Dilatation of the right chambers* of the heart in its *primary* form may be due to :

- (a) Fatty deposit.
- (b) Fatty degeneration.

(c) Inflammatory softening.

(d) Fibroid conversion.

In the *secondary* form it may result from :

(a) Obstruction in the lungs.

(b) Obstruction or regurgitation at the mitral orifice.

(c) Obstruction or regurgitation at the aortic orifice.

In the primary form, dilatation of the right ventricle is usually associated with thinning of its walls; but when secondary to any of the causes indicated in the latter category, it is preceded by hypertrophy, provided that general nutrition be unimpaired; hence there is thickening of the walls with enlargement of the cavity. But, as already stated, hypertrophy of the right ventricle, even in a moderate degree, is rare.

The differential diagnosis, from direct physical evidence, of dilatation of the right ventricle, accompanied by, or unattended with, thickening of its walls, is not practicable in the present state of medical knowledge; but dilatation of some form having been determined, the history and collateral signs actually existing will enable the physician to form a presumptive judgment almost equivalent to a positive diagnosis.

This difficulty arises from the twofold circumstance, that the right ventricle is shielded from the touch by the sternum, and that the factor contributed by it to the first sound is so insignificant, that even when exaggerated, it is not distinctly appreciable.

Dilatation of the right ventricle can scarcely be presumed to exist without a corresponding state of the right auricle, owing to the invariable accompaniment of expansion of the tricuspid orifice. The right auricle may, however, be dilated without accompanying dilatation of the right ventricle; when, for example, organic obstruction exists at the tricuspid opening, without a corresponding condition of the mitral orifice, a very rare occurrence, the right auricle would alone undergo hypertrophy and dilatation.

Dilatation of the right ventricle may be diagnosed by extension of precordial dulness to the right, unaccompanied by displacement of the apex to the left side, and by the existence of jugular pulsation synchronous with ventricular systole. This

latter sign, first pointed out by Lancisi, but by him, and subsequently by Laennec, erroneously interpreted as evidence of hypertrophy of the right ventricle, is really diagnostic, not of dilatation of the right ventricle, but of tricuspid inadequacy as shown by Morgagni. This latter condition is, however, rarely met with, except as the result of dilatation of the right ventricle, which may be, therefore, likewise presumed to exist. Hope regarded this sign as evidence of hypertrophy with dilatation of the right ventricle; but Bertin correctly interpreted it as a sign of dilatation only of that chamber, with a corresponding condition of the right auriculo-ventricular opening, and consequent inadequacy of the tricuspid valve.

Morgagni drew a just distinction between jugular pulsation resulting from simple auricular reflux, and that from ventricular regurgitation. In the former case, as he remarks, the pulsation of the jugular vein observes no relationship in time to the systole of the ventricle; whereas, in the latter, it is strictly synchronous with the pulse, and therefore with the contraction of the ventricle.\*

This distinction is, as I believe, well founded. A tremulous movement of the jugular veins, extending over the entire period of ventricular diastole, and corresponding to the feeble undulatory contractions of the auricle prior to its final and abrupt presystolic contraction, is of frequent occurrence, and represents a minor degree of congestion of the right side of the heart. I have, however, repeatedly observed both kinds of jugular movement in the same case; viz., a full reflux wave synchronous with the contraction of the ventricle, and a feeble and continuous movement immediately succeeding it: both being readily arrested by light finger-pressure above the clavicle.

Longet, adopting the views of Potain, represents the jugular veins as normally exhibiting the following series of movements; viz., two abrupt elevations in quick succession; the former due to the arrest or retardation of the entrance of blood into the auricle at the moment of its contraction, and the latter, to the retrograde shock or pressure arising from the closure of the tricuspid valve, or the lateral pressure of the great arteries upon

\* *Seats and Causes of Disease*, translated by Cooke, 1822, vol. i., p. 377.



the adjacent veins at the moment of their expansion ; perhaps to both these causes combined. Alternately with the twofold expansion of the vein just described, may be noticed a partial subsidence or sinking of it, due to the successive diastole of the auricle and the ventricle ; and, finally, a slow movement of expansion from the refilling of the vein from the periphery.\*

The annexed figures (XXXII. and XXXIII.) of Friedreich, copied from Dr. Gee's work,† represent abnormal venous pulsation. Figure XXXII. shows *systolic* dicotism of the jugular

FIG. XXXII.

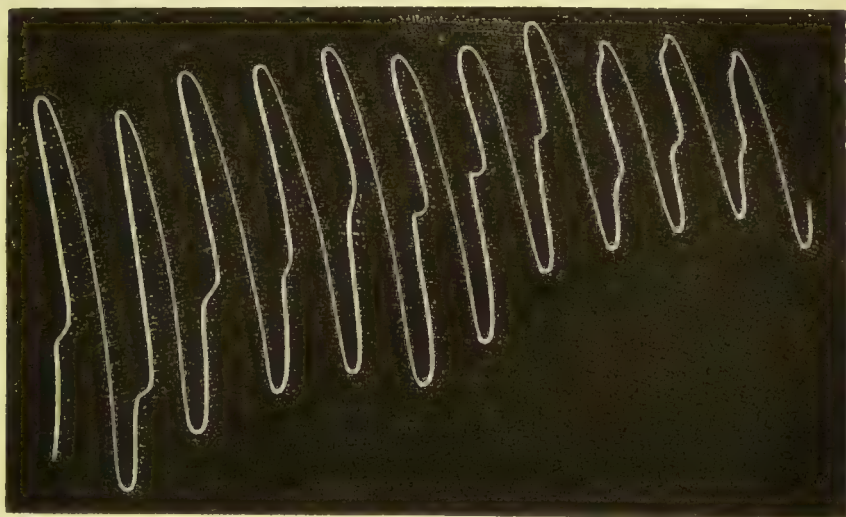
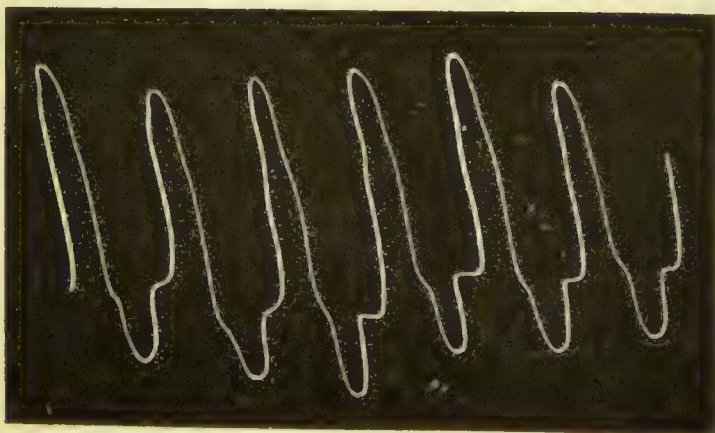


FIG. XXXIII.



\* *Traité de Physiologie*, 1869, tom. deuxième, p. 250.

† *Auscultation and Percussion*, 1870, p. 167.

pulse, the first and lesser portion of the wave of ascent being the result of auricular contraction, the second and larger, that of ventricular systole. Figure XXXIII. represents a phenomenon of occasional occurrence, namely, *diastolic* dirotism of the jugular pulse. This is the result of persistent repletion of the right auricle, and consequent sudden arrest, towards the end of diastole, of the further entrance of blood.

Dilatation with thinning of the right ventricle, from softening, whether fatty or inflammatory; and dilatation with hypertrophy from emphysema and recurrent bronchitis, or other chronic obstruction to the pulmonary circulation, are of less unfavourable augury than the corresponding changes in the left ventricle; because of the much less liability to rupture of a softened right ventricle than of a left ventricle similarly affected, and because chronic obstruction to the circulation in the lungs is usually of a remittent character. There is, however, considerable danger from thrombosis of the right ventricle and pulmonary artery.

*Dilatation of the right auricle* is usually associated with, and consecutive to dilatation of the right ventricle, owing to the facility with which, in protracted engorgement of the right ventricle, the tricuspid orifice becomes expanded, and its valve thereby inadequate. It may exist, however, as already stated, independently of any change in the capacity of the right ventricle, where tricuspid narrowing is the only valvular lesion present; but this is a pathological unity of extreme rarity, and separate dilatation of the right auricle is, therefore, very seldom witnessed.

A right auricle dilated in a high degree may simulate aneurism of the ascending aorta. The most remarkable example of this kind on record is given by Dr. Stokes. A man, past middle age, was admitted into the Meath Hospital suffering from symptoms of cardiac disease. The heart pulsated in its natural position; its action was feeble and irregular. There was dulness to the right of the sternum, extending from the second to the fifth rib, and to an equal extent in this situation an expansile pulsation was felt resembling that of an aneurism, and synchronous with the first sound of the heart. No note was taken as to whether murmur existed. In regard to aneurism, a negative

diagnosis was made, chiefly from the coexistence of irregular action of the heart. The patient sank from congestion of the lungs. The aorta was healthy; the lungs were emphysematous and congested. The right ventricle was dilated and hypertrophied, and the right auricle "resembled a vast purple tumor which concealed the whole of the anterior portion of the right lung. Its parietes were in many places extremely thin, while in others the fleshy columns, especially in the appendix, were hypertrophied. Its cavity contained more than a pound of fluid but grumous blood."\* The pulsation of the auricle was here due, as Dr. Stokes remarks, to systolic reflux from the ventricle. The difficulty of differential diagnosis from aneurism must have been very considerable in this case, and in the enumeration of possible sources of error, the example, though unique, should not be lost sight of. The necessary coexistence, however, of pulmonary and general venous engorgement must have aided considerably towards a right conclusion.

Where the right side of the heart is dilated and engorged, whether from debility of the walls or from primary obstruction in the lungs, I would not hesitate to abstract blood locally, or even generally, but with circumspection. The application of a few leeches to the precordium, or the abstraction of a few ounces of blood by cupping over the lungs where they are the seat of primary obstruction, the use of the hot foot-bath and a mild saline aperient, are usually followed by great relief to the patient. Where, in the absence of valvular disease on the left side, general venous congestion amounts to a state of cyanosis, with great dyspnœa, distention of the cervical veins, irregular pulse, and feeble, but tumultuous action of the heart, I would not hesitate to bleed from the arm to ten or twelve ounces. Medically, I consider digitalis, when the stomach will tolerate it, the most valuable agent we possess in the treatment of weak and engorged right heart. I prescribe it in doses of  $\mathfrak{m}_x$  of the tincture, with an equal quantity of chloric ether and of tincture of perchloride of iron, in an ounce of water every third hour. I have never witnessed from its use more unpleasant results than

\* *The Diseases of the Heart*, p. 274-5.



occasional sickness of stomach ; but when this occurs the medicine should be stopped for at least twenty-four hours. It increases the contractile force of the heart, slowing its action, and restoring its regularity of function. Thus, by its action, when it is administered in well selected cases, the strength and volume of the radial pulse are increased, and its regularity, if absent, is restored, whilst the renal secretion is augmented. The allowance of liquids should be very limited, and alcoholic stimulants should be given in full quantity.

Dilatation of the left, like that of the right auricle, when it exists separately, depends upon contraction of the auriculo-ventricular opening of that side, and is associated with thickening of its walls. It must, however, ultimately give rise to dilatation of the right chambers, and should, therefore, be regarded as representing an early stage of a process which must issue in more general dilatation of the heart.

From the anatomical position of the left auricle, dilatation of this chamber must be difficult of recognition by independent signs ; but, in conjunction with those of mitral narrowing, the existence of continuous feeble undulatory pulsation in the second and third intercostal spaces of the left side, about one inch from the sternum, may be regarded as evidence of this condition.

Separate dilatation of the left, like that of the right ventricle, may be primary, and dependent upon typhoid, inflammatory, or fatty softening ; or it may be secondary to organic disease at the mitral orifice, involving valvular inadequacy and regurgitation, or to disease at the orifice of the aorta giving rise to obstruction, regurgitation, or both, provided there be softening of the walls.

Softening of whatever kind, if primary, is followed by dilatation with thinning of the walls, when time has elapsed sufficient for the complete development of the pathological series.

The symptoms are weak and failing pulse, which is, likewise, usually quick, intermittent, and irregular. It may, however, be abnormally slow and large in the fatty form of softening, but here the radial artery is usually tortuous, and not unfrequently rigid. Vertigo is of frequent occurrence, and there is a tendency to syncope on suddenly assuming the erect posture, and on



making unusual effort. In cases of softened and dilated left ventricle, the apex-point, as determined by the sounds, is displaced to the left, and the apex-beat is feeble and diffused. The first sound is weak, but short and high pitched; the impulse is either in the last degree feeble, or altogether inappreciable. At the base the second sound is sharp, but weak, and if the aorta be not rigid by atheromatous or calcareous change, it is but faintly audible at the right second costal cartilage. In the treatment of typhoid softening, as pointed out by Dr. Stokes, wine in full quantity constitutes the remedy; under its use the pulse usually becomes slow at the same time that it acquires volume and strength. Alcohol is also indispensable in the other two forms of softening of the left ventricle, and in all three I prescribe quinine, strychnine, and iron.

When dilatation of the left ventricle is the result of valvular lesion it is always accompanied by thickening of the walls. The diagnosis and the treatment will be considered in connexion with the valvular lesion upon which the dilatation depends.

The following are examples of dilatation of the heart.

CASE XXXII.—*Dilatation of the Heart and Resolution of the First Sound.*

Catherine M., aged fifty-five years, a domestic servant, came under my notice as an extern patient of the hospital, March 12th, 1869. Had been for some time previously suffering from flatulence and sensations of "sinking" at the stomach, vertigo, and occasional faintness. Had never spat blood. Was pale and thin, but free from cough; pulse 96, weak, and occasionally intermittent. Sounds of heart morbidly clear over precordium generally; but, at the apex, the first sound was resolved into its two elements of impulsion and valve-click, of which the former was dull and soft, whilst the latter, which immediately succeeded, was sharp and "valvular" in character. The former, by its rhythm, might be mistaken for a presystolic murmur, but was readily distinguishable from it by the soft character of the sound, and by its coincidence with the cardiac impulse. R. Tincture of digitalis and chloric ether, of each ℥v thrice daily. Not seen again.

Although I incline to associate reduplication or resolution of the first sound at the apex with simple hypertrophy of the left ventricle as the usual attendant condition, I would not be understood to deny its occasional occurrence in connexion with other organic changes of the heart; for example, thickening, with some degree of dilatation, of the left ventricle.

CASE XXXIII.—*Dilatation and rapid Action of the Heart; Irregularity and want of Correspondence between the Action of the Heart and the Radial Pulse.*

Margaret L., a widow, aged forty-five years, visited the hospital as an out-patient in February, 1868. Twelve years previously she had swelling of the legs after cold caught in her first confinement. She is remarkably nervous and fretful; pulse at wrist 108, so weak as to be with difficulty counted, and arhythmically irregular and intermittent; heart acting at rate of 192, irregular, but not intermittent, its sounds sharp and clear, and accompanied by a musical note not in harmony with either sound; apex-beat half an inch external to the nipple. Feels weak occasionally after an effort, and is sometimes faintish after rising from bed, so that she is obliged to lie down again, but has never fainted; breathing on these occasions much accelerated. To have aperient pills; also tincture of iron and chloric ether, of each 3ij, with infusion of quassia to 3vij: a tablespoonful to be taken thrice daily.

Admitted into hospital, April 27th. Heart acting at rate of 182; sounds clear and ringing, but unaccompanied by murmur. Pulse weak, irregular, and intermittent, and, as to rate, out of harmony with the action of the heart. To have tincture of digitalis, tincture of the American wild cherry (*Prunus Virginiana*), and chloric ether, of each ℥v, every fourth hour.

May 7th. Pulse and heart-rate correspond, 96 per minute with slight irregularity. No sensation of sinking; can sleep and take food well; heart-sounds less clear and sharp. Discharged.

June 17th. Pulse weak, irregular, and intermittent, 168, and corresponding with action of heart. To resume last medicine.

April 2nd, 1869. She again presented herself. Pulse 96,

with an uncertain number of abortive beats; action of heart varies from 144 to 168; sounds clear. There are, occasionally, a few strong and regular beats; these are represented in the radial pulse, and are succeeded by an uncertain number of weak pulsations in rapid succession, which are not fully, or only by abortive pulsations, represented in the radial pulse. She suffers from dizziness, and is in danger of falling after exertion, especially that of ascending a flight of stairs.

The musical note audible at the precordium on the occasion of the first visit, but not subsequently, was of gastric origin. I have repeatedly heard this sound in connexion with gastric tympany, and have had no difficulty in identifying its source by its metallic character; its want of *strict* synchronism with either sound; its capriciousness, or occasional absence during one or more cycles of cardiac movement without any intelligible cause; and, finally, by the existence in the precordium of tympanitic percussion-resonance continuous with that of the epigastrium.

The beneficial action of digitalis is exemplified in the comparative regularity, increased force, and reduced rate of the heart's action, and in the general improvement of the patient under its use in May, 1868.

CASE XXXIV.—*Hypertrophied, Dilated, and Fatty Heart; "Ascending and Descending" Respiration; Sudden Death.*

Joseph C., aged sixty-two, an educated man of gigantic stature, presented himself as an extern patient, May 12th, 1869. He stated that during the preceding fifteen years he had suffered from shortness of breath, with occasional attacks of bronchitis, and that a fortnight previously he had spat some blood. There was no œdema; the pulse was 96, arhythmically irregular, and intermitting. He was subject to sudden accessions of dyspnœa, with tendency to fainting, which usually came on in bed, and obliged him to rise promptly in order to catch his breath. Had not been able to lie down for the preceding week, owing to the immediate accession of attacks of this kind on his attempting to do so. To have tincture of digitalis, tincture of perchloride of iron, and chloric ether, of each ten drops, thrice daily.

May 14th. Pulse 96, intermitting; feet swollen; heart's action irregular and intermittent; its sounds clear, but weak, and free from murmur. Respiration "ascending and descending" at intervals, and then his sight is dim. Declares he feels better, and has been able to get some sleep since he began to take the medicine.

19th. Admitted into hospital. There is now great œdema of the lower extremities, and dyspnœa threatening asphyxia; pulse intermitting; breathing "ascending and descending," or exhibiting alternations of gradual increase and decrease of rate, succeeded by a brief period of complete apnœa. To have previous medicine.

20th. Slept well in recumbent posture last night, and declares he feels infinitely better.

23. No return of dyspnœa. Sleeps well; œdema disappearing; attributes his improvement to the medicine, which he calls his "preservative," and demands to have it repeated. Passed since yesterday about a gallon of pale urine.

This man was discharged at his own request a few days afterwards, feeling well and free from dyspnœa, the œdema having entirely disappeared. The pulse, however, continued irregular.

July 16th. Called on me at my residence to-day. Had an aggravated attack of dyspnœa last night, causing him to start out of bed to save himself from suffocation. Is very thin; pulse irregular and failing; great œdema of feet and legs; kidneys and bowels acting well. No cardiac impulse to be felt, but a general and feeble movement is perceptible to the hand placed on the precordium. At the apex and in the line of the aorta both sounds were remarkably obscure, whilst at the ensiform cartilage they were morbidly clear. No hæmoptysis. To have, thrice daily, ℞vj of the tinctures of digitalis and iron, with chloric ether, as previously.

17th. Visited at his lodgings. Passed a better night, and reported much improved.

August 8th. I discontinued my attendance, the patient being in all respects improved, able to sleep in the recumbent posture, appetite fairly good, œdema having disappeared; but the action and sounds of the heart and the radial pulse still were as above noted.



A few weeks subsequently to the last mentioned date, I learned, through the kindness of Dr. Dudley White, surgeon to the city coroner, that this man had died suddenly at his lodgings. Dr. White examined the body at the coroner's inquest, and informed me that the heart was large and fatty; but no more special report was made.

The advantage derived from the use of digitalis, even where the heart is fatty, is here strikingly exemplified.

CASE XXXV.—*Dilated and Fatty Heart.*

Patrick D., aged sixty years, a labourer, temperate, and never had rheumatism, was admitted into hospital, June 12th, 1868. Nine weeks previously his breathing became short after a wetting, and a month later his feet began to swell. Respiration 36; pulse 130, and irregular; tongue coated, bowels constipated and flatulent; well marked arcus senilis.

The urine was acid, sp. gr. 1.025, and free from albumen; feet cedematous and congested. Muco-crepitus with inspiration over apex of left lung, and here respiration was feeble, but elsewhere it was normal; slight jugular pulsation of inspiratory rhythm; precordial dulness normal; both sounds of heart sharp and clear at the apex and at the base; action of heart irregular, but no cardiac murmur to be heard.

June 15th. Pulse 120, irregular; great increase of urine.

16th. Pulse 84, irregular; heart-sounds sharp and clear; urine, about three quarts daily. Discharged improved, and not subsequently heard of.

CASE XXXVI.—*Weak and Dilated Left Ventricle in a gouty subject; Postsystolic Apex-murmur; Cessation of all Morbid Signs under Treatment.*

Mr. W., aged fifty-six years, unmarried, and temperate, holding a responsible position in the Civil Service, requiring nine hours of office duty daily, consulted me, March 15th, 1872. Five years previously, after a fainting fit in church, he consulted an eminent physician, whose opinion, as reported by him, was that he had valvular disease of the heart. Another physician of no less eminence, subsequently expressed a similar opinion.

He was well nourished, could take exercise with enjoyment, and felt himself improved by it; had no cough or derangement of respiration, and no œdema; had never spat blood. Pulse 96, small, regular, and equal, but occasionally intermitting; apex-beat in nipple-line and fifth intercostal space, and of moderate force. Both sounds were here distinct and normal; the first, however, was sharper than natural, and was succeeded by a faint blowing murmur (postsystolic), extending quite up to the second sound, but inaudible elsewhere than at the apex. Both sounds were a little more sharp than normal at the base, and the second sound was not accentuated in the pulmonary artery,

This gentleman had had rheumatic fever in childhood; his habits were irregular in regard to diet, no food whatever having been usually taken between breakfast at 9, A.M., and dinner at half-past 6, P.M. He declared that he had been rather improving in health during the last five years, and he desired to have a definite opinion as to the state of his heart, with a view to claiming a retiring allowance under the regulations of his department.

I gave a confident opinion that no valvular disease existed; that the symptoms and signs referable to the heart were due to weakness and dilatation exclusively; and that, under treatment they would entirely disappear. I added, that his illness was, in my judgment, connected with latent gout. To have ℞ of tincture of digitalis, with ℞ of tincture of perchloride of iron, and ℞ij of tincture of colchicum and spirit of chloroform thrice daily. To have every night, with a view to correction of flatulency with which he was much troubled, gr. j of blue pill, with gr. j of compound rhubarb pill, and grs. ij of dried soda. He was directed to take one or two glasses of good dry sherry daily, and to have some luncheon at mid-day.

I have since repeatedly seen this gentleman; he had continued the use of the medicine above mentioned for several months, and was when I last examined him (September, 1872) in excellent health, and quite free from all morbid signs referable to the heart. The murmur which existed at the apex on the occasion of the first visit, ceased within a few weeks.

It would be presumption in me to attempt to explain why the two deservedly eminent men who had diagnosed valvular lesion,

had arrived at that conclusion. I may, however, be pardoned the suspicion that the error into which they undoubtedly fell, was due to the existence of a systolic murmur, having its origin at the apex, and associated with rheumatic antecedents. A mitral murmur of systolic or postsystolic rhythm, is even still, by most stethoscopists, regarded as *prima facie* evidence of organic lesion of the mitral valve. In a previous Chapter (page 282), I have endeavoured to show that reflux murmur at the mitral orifice may exist as a result of weakness and excentric yielding of a portion of the wall of the left ventricle, at the acme of systole, and without structural alteration of the mitral valve. I believe the case now under consideration was an example of this kind; and the cessation of the murmur coincidently with the general improvement of the patient under tonic treatment, may, I think, be legitimately offered as evidence of the correctness of that opinion.

CASE XXXVII.—*Weak and Dilated Left Ventricle; Non-organic Mitral Murmur.*

Frances C., aged fifty-eight years, the mother of eight grown children, became an out-patient of the hospital in the autumn of 1865. She had worked hard in early life, and twenty years prior to her visit, she began to suffer from weakness and palpitation on making unusual exertion; latterly she had been subject to intercurrent attacks of bronchitis. In the spring of 1867 she felt more than ordinarily weak, and experienced lightness in the head after quick movement or exertion of any kind. In the course of the preceding winter she had a slight attack of rheumatism, in which the right elbow, knee, and ankle were swollen, but there was no complaint of pain in the left side during this attack. Pulse 84, and in an extreme degree arhythmically irregular; it likewise occasionally intermitted twice or thrice in succession, sometimes only once. The radial arteries were tortuous, and pulsated visibly. Respiration regular, 30 in the minute, and unembarrassed. No venous turgescence of the neck, or visible pulsation of the carotid or temporal arteries. Precordial dulness normal in extent. Cardiac impulse weak, and apex-pulsation slightly to left of usual position. The first

sound was accompanied by a faint murmur at the apex, and the second, at midsternum, was likewise accompanied by a feeble murmur, which seemed to constitute the initial portion of the sound, the terminal portion of which was normal. Distinguished from murmur, the sounds were remarkably sharp and clear. Tongue clean, bowels confined. To have aperient pills, and ammonia and chloric ether in camphor water, also a moderate allowance of wine. She was subsequently directed to take ℥v of liquor strychniæ, in a bitter infusion, thrice daily.

July 26th. Reported that she can now rise at 5 o'clock, A.M., a feat previously impossible. Pulse still irregular and 84, but occasionally slower, owing to the occurrence at intervals of an abortive pulsation not represented in the radial pulse. *No cardiac murmur now to be heard.* Medicine continued.

November 19th. Feels weak, action of heart and radial pulse arhythmically irregular and intermitting; heart-sounds clear, and no cardiac murmur.

April 5th, 1869. Pulse 96, still occasionally irregular; she feels very well, and is able to work.

June 12th. Slight œdema of face; action of heart and pulse arhythmically irregular and intermitting; looks well, but complains of uneasy sensations in region of heart, with a distinct perception of its intermissions. To have tincture of the perchloride of iron, chloric ether, and tincture of digitalis, of each ℥x in an ounce of water, thrice daily. Having, at this time, ceased to conduct the extern department of the hospital, I lost sight of this patient, but I have since repeatedly seen her in the streets, and to all appearance in her usual health.

The diagnosis of mitral reflux by yielding of the walls of an already weakened and dilated left ventricle, and without valvular lesion, was made at the first interview with the patient in 1867. The subsequent cessation of the murmur under tonic treatment showed that this view of its origin was correct. The occurrence, at one period, of a faint murmur at the orifice of the aorta, prefacing the second sound, subsequently ceasing, and **not** returning, is explicable only on the assumption of a shred of fibrin attached for the time to the free edge of the valve, and rendering it partially inadequate at the moment of closure.



*Atrophy of the heart* must be regarded as the opposite of its enlargement. Under the various forms of hypertrophy and dilatation, all possible conditions of the organ, in regard to thickening of its walls and dilatation of its chambers, have been considered. Most, if not all these conditions imply enlargement of the heart, the only *theoretical* exception being dilatation with thinning of the walls,\* but, even here, I am convinced there is absolute enlargement in nearly every instance, from the preponderance of dilatation over parietal thinning.

Bouillaud understands by atrophy the opposite of hypertrophy, and recognizes three forms of it corresponding to the three forms of the opposite condition, hypertrophy; viz., *simple atrophy*, in which the walls are attenuated and the cavities normal as to capacity; *excentric atrophy*, or attenuation of the walls, with dilatation of the cavities; and *concentric atrophy*, the walls being normal or increased in thickness, and the cavities reduced in capacity.

Bertin makes a similar classification, but under different designations. The heart must be, however, in every instance below the normal standard as to volume, according to him.

Having reference to the general reduction in volume of the heart necessary to constitute a state of atrophy, I think a preferable, because a more natural, classification would be the following: *simple atrophy*, consisting in a general reduction in volume of the heart, the walls and cavities bearing a normal proportion to one another and to the size of the organ; *excentric atrophy*, in which, whilst the heart is below the normal size, the walls are thinned and the cavities dilated relatively to the walls; *concentric atrophy*, in which, the heart being in volume less than normal, the walls are relatively thickened and the cavities reduced in capacity.

Rokitansky enumerates the following causes of atrophy of the heart; viz., general marasmus from typhus, tuberculosis, or the cancerous diathesis; the pressure of secondary products in the mediastinum; pericarditis; fatty accumulations on the heart; and contraction of the openings of the coronary arteries. The

\* I purposely omit so called "concentric" hypertrophy, the existence of which, as a pathological condition, I cannot recognize.

tissue of the organ may, he says, be either reddish-brown and tough, or fawn-coloured and easily torn.\* As stated by Bertin, atrophy of the heart is due to diseases other than cardiac; but there is at least one exception, namely, where atrophy depends upon adhesion of the pericardium.

Doctor Stokes is of opinion that atrophy, quite as often as hypertrophy, is found associated with adhesion of the pericardium, and he surmises that the former condition is due to pressure upon, and obliteration more or less complete of, the coronary arteries, by the contraction and pressure of the false membrane. He further states, on the authority of Professor R. W. Smith, that calcification of the pericardium is, when present, invariably associated with atrophy of the heart.†

Barlow‡ and Cheevers§ have maintained that obliteration of the pericardium by adhesion is more frequently followed by atrophy than by hypertrophy of the heart.||

Doctor Walshe, whilst holding that hypertrophy is the ordinary change of the heart consecutive to adhesion of the pericardium, admits that he has met with at least one example of atrophy of the left ventricle from the constricting action of an enveloping false membrane. He attributes the wasting in this instance to pressure upon the coronary artery.¶

The most frequent causes of atrophy of the heart are undoubtedly diathetic diseases of debility, and, *par excellence*, tuberculosis and cancer, as first pointed out by Louis and Bizot. The heart in such case is pale, but firm, and structurally sound, except in so far as it may be the seat of tubercular, cancerous, or other adventitious deposit; and its walls and cavities are in normal relative proportion.

I have not seen atrophy from accumulation of fat upon the heart, and pressure therefrom on the coronary arteries, as mentioned by Flint.\*\* By atrophy I understand reduction in the

\* *Pathological Anatomy*, Sydenham Society's edition, vol. iv., p. 170.

† *Opus citat.*, p. 12.

‡ *Gulstonian Lectures*, 1843.

§ *Guy's Hospital Reports*, vol. ix.

|| *Vide antea*, p. 347.

¶ *The Diseases of the Heart*, p. 274.

\*\* *Opus citat.*, second edition, 1870, p. 92.

total volume of the heart. In the form mentioned, however, there is no diminution of volume, the proper substance of the heart is only in part replaced by an adventitious structure. Atrophy of the heart may be due to atheromatous change in the coronary arteries, or to a similar affection of the aorta obstructing the entrance to these vessels. In such case it would be associated with retrogressive change of structure, granular or fatty, and the heart would be likewise soft and lacerable, and of a dark fawn or a light brick-colour. It may be likewise congenital, the viscera generally, and the body itself being similarly undeveloped. The heart, therefore, would be in due proportion to the remainder of the body, and its walls and cavities strictly proportionate. Many hold that atrophy of the heart may be the result of general senile decay; but Cruveillier denies atrophy of the organic muscles in the aged, whilst admitting that of the muscles of animal life.\* In this opinion I entirely concur. Wasting of the organs essential to life, in the absence of specific disease, is not an ordinary accompaniment of advancing years. In acute muscular atrophy, the voluntary muscles are usually alone affected.

There is, however, at least one case on record, in which the heart participated in the wasting and degeneration of this disease. The patient was a female, aged twenty-six years, under the care of Dr. Headlam Greenhow. She died of wasting palsy, which was characterized by the usual symptoms, within a period of less than five weeks. The muscles of the forearms, the erectors of the spine, the pectorals, and the diaphragm, were wasted, and had undergone the diffuse granular change usually exhibited by certain of the voluntary muscles in this disease. The heart was pale and somewhat fatty on the surface, and its muscular structure, especially that of the right ventricle, exhibited well pronounced granular degeneration, with some interfibrillary fat.†

Chronic pericarditis, with general adhesion of the pericardium, may be the cause of atrophy of the heart. I have already ex-

\* *Anatomie Pathologique*, l. xxx.

† *Clinical Society's Transactions*, vol. vi., 1873.

pressed the opinion (p. 368) that hypertrophy is the ordinary result of pericarditis with adhesion, occurring at a period of life when general nutrition is active, *i.e.*, in childhood and youth, provided the health of the patient be otherwise good, and no special impediment to the nutrition of the heart exists, such as obstruction of the coronary arteries. In the opposite circumstances, that is, where pericarditis occurs in a person of feeble nutrition, whether from advanced age or broken health, or where it is preceded or accompanied by obstructed coronary circulation, the result is likely to be atrophy, and most probably that of the degenerative kind.

Chronic effusion into the left pleural cavity may, by the pressure exercised upon the heart, and the general impairment of nutrition to which it usually gives rise, be the cause of atrophy of the heart. Dr. Flint gives an example of this kind in an adult female. The heart was flabby, fawn-coloured, and probably fatty; it weighed only four and a-quarter ounces, and the walls of the left ventricle were less than a-quarter of an inch thick.\*

The "pigmentary atrophy" of Reynaud† is usually met with in combination with fatty degeneration, and is, I believe, a form of that change, but modified by the accident of pigmentation. In this condition the section of the heart exhibits a bronzed or slate colour, and in the fibres pigment granules are observed in addition to oil dots.

The pulse in all cases of atrophy of the heart is small, quick, and, it may be, all but inappreciable; but, in those forms in which it is unassociated with fatty or other tissue-change in an advanced stage, it is regular. In the latter, however, the pulse is irregular, intermittent, and failing, and syncope is of frequent occurrence. The area of precordial dulness, both deep and superficial, is contracted, and the cardiac impulse is limited and feeble. The sounds are sharp and clear where the structure of the heart is sound; but where it is softened, the first sound is dull, faint, and scarcely audible. Flint is certainly in error when he states that the sounds are "abnormally feeble or inappreciable," irrespec-

\* *Opus citat.*, p. 93.

† *Nouveau Dictionnaire de Médecine et Chirurgie*, 1868.



tively of the structural condition of the heart. In infants, with still smaller hearts than those met with in most examples of atrophy, the sounds are remarkably clear and distinct, even *in utero*.

Atrophy will be exemplified in the following cases.

CASE XXXVIII.—*Atrophy with General Dilatation of the Heart.*

Bridget M., aged sixty-six years, pale and delicate looking, visited the Mater Misericordiæ Hospital as an out-patient in 1867. She was then suffering from palpitation, the action of the heart and the radial pulse being arhythmically irregular and intermittent; there was also a tendency to syncope, and partial but temporary failure of vision. Both sounds of the heart were equally clear, sharp, and distinct. She was relieved by the use of iron, digitalis, and chloric ether. She again presented herself in May, 1870, complaining of a hard and painful tumor in the left axilla, which was manifestly malignant; she then suffered also from great weakness and from the former tendency to syncope. The cardiac rhythm was deranged, the impulse being both arhythmically irregular and unequal. Thus, there would be two or three beats in rapid succession, but at unequal intervals, and at a rate of more than 100 in the minute, and then a long intermission both in the impulse of the heart and in the radial pulse would occur. The sounds of the heart were very clear and sharp. I lost sight of her after this date.

CASE XXXIX.—*Hepatic Symptoms with Dropsical Effusion into the Peritoneum; Variation in the Volume of the Liver, and Enlargement of the Spleen; Paracentesis Abdominis, and Peritonitis; Irregularity of the Heart's Action, and Muffled First Sound; Anasarca and Hydrothorax; Paracentesis Thoracis; Death. Chronic Pericarditis with General Dense Adhesion and Calcification; Excentric Atrophy of the Heart; Hydrothorax with Carnification of the Lungs; Cirrhosis of the Liver; Enlargement of the Spleen and Kidneys; Chronic Peritonitis.*

James R., a young man of good education, aged nineteen; was

admitted into the Mater Misericordiæ Hospital in June, 1863, suffering from uneasy sensations, rather than pain, in the region of the liver. There was ascites, and likewise œdema of the left lower extremities; pulse small, weak, and variable as to rate, but regular. Paracentesis abdominis was performed, and nine gallons of serum were removed. Two months subsequently he had improved so much in health that he was able to leave hospital. There was then no ascites, and no enlargement of the liver could be detected.

Five months later the liver was found enlarged, and irregular on the surface, and there was slight effusion of serum into the peritoneum. The pulse at this date was full, strong, and regular. General health good.

One month subsequently there was jaundice which lasted two days; and two months later still, viz., in May, 1864, he had so much improved in health that he had gained flesh. Pulse 102, and full; liver much enlarged and thickened, but smooth on the surface.

In August there was again liquid accumulation, but only to a slight amount, in the abdomen; the liver was tender to pressure, and the left leg was œdematous.

From this date till April, 1868, when he was readmitted into hospital, the patient presented himself at intervals of a few months. It is unnecessary here to give details as to his varying condition during this lengthened period. These I have elsewhere published at length.\* I shall give such portions only as are relevant to the present subject.

On the 31st May, 1866, a note was for the first time made that the action of the heart intermitted.

On the 13th July, 1867, more than a year later, the further observation was recorded, that "The first sound of the heart was masked, the second sound being exceedingly clear, and the heart still irregular in action, and intermitting."

Under date 15th November following, it is noted that "both sounds were remarkably clear, and of a ringing metallic character."

There were recurrent attacks of jaundice, which usually lasted

\* *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., 1866, p. 281.

not more than a few days; the spleen became perceptibly enlarged; serous effusion now took place into the cavities of the right pleura and the pericardium, but very slowly, and the quantity present varied from month to month, inversely as the action of the kidneys. The ascites, however, steadily increased, and, concurrently, the patient lost flesh and became weaker; the lower extremities and genitals became so swollen that it was found necessary to puncture them in order to avoid gangrene, and enable the patient to micturate.

On the 19th March, 1868, both sounds of the heart, as heard beneath the lower sternum, were clear and ringing as before, but the action was irregular and intermitting; urine, sp. gr. 1.018, acid, and containing lithates and a trace of albumen.

On the 25th April the right pleural cavity was full of liquid, the cervical veins were engorged, the face was livid, and respiration was so much embarrassed that death from asphyxia seemed imminent. Paracentesis thoracis was performed, and shortly afterwards the patient sank exhausted.

Both lungs were found congested, and the base of the right carnified. The abdominal cavity was full of dark serum; the viscera were universally adherent by dense false membrane, which enveloped the liver in a thick envelope. In the recesses between the agglutinated intestines small pools of serum were found, which glistened with crystals of cholesterine. The liver was in an advanced state of cirrhotic and fatty change. The spleen and kidneys were greatly enlarged and congested, but structurally healthy.

The pericardium was calcified, greatly thickened, and closely and firmly adherent to the heart. It presented a cartilaginous appearance in most parts; but in certain situations it was calcified, and resisted the knife; and in a few places on the right side, and to a less extent on the posterior left aspect of the left ventricle, there were large masses of gritty calcareous matter, partly imbedded in the pericardium, and partly in the substance of the heart.

The vena cava superior and the vena azygos were considerably dilated, showing the obstruction to which the abdominal venous circulation had been for a long time subjected. The

heart was much reduced in size, soft, and flabby. The right cavities were dilated, and their walls remarkably attenuated. The tricuspid orifice was large, and both auricle and ventricle were occupied by masses of decolorized fibrin, connected through the tricuspid opening and extending into the pulmonary artery. The left auricle was greatly dilated, and the left ventricle was dilated, and its walls soft and greatly thinned. There was no valvular disease whatever. The substance of the heart was of a dark fawn colour, but not fatty.

It is not improbable that at the date of the first observation of intermittence of the heart, viz., in May, 1866, latent pericarditis existed; and in July of the following year the muffled character of the first sound may have been due to liquid effusion into the pericardium, which was subsequently absorbed, and followed by adhesion and calcareous conversion of the adventitious matter.

I conclude that in the foregoing case atrophy of the heart was due to the twofold cause of general malnutrition and special interference with the nutrition of the heart, arising from the close adhesion of the pericardium. Calcification of the adventitious connective tissue was a subsequent event, and likewise an example of atrophic degeneration with deposition of calcareous particles.

CASE XL.—*Scirrhus of the Mesentery; Death by Peritonitis from Sloughing of the Intestines and Effusion of their Contents. General Adhesion and Calcification of the Pericardium; Simple Atrophy and Granular Degeneration of the Heart.*

A poor man named P., a native of North Wales, aged forty-three years, was admitted into hospital on the 26th November, 1872, on the recommendation of my friend Dr. Roberts of Portmadoc. He had complained for six months previously of occasional dull pain in the abdomen, and irregularity of the bowels, and, when admitted, he was much emaciated. He suffered from dull remittent pain in the hypogastrium, which was aggravated whenever the bowels were confined; a hard, rugged, and movable tumor was detected in the right iliac fossa; there was



occasional diarrhoea, the stools being free from faecal odour, and resembling liquid mortar. The pulse was quick and small, but regular, precordial dulness limited in extent, and cardiac impulse remarkably feeble; the sounds were weak but clear, and unattended with murmur. A few days before his death, which took place on the 31st December, the abdomen became inflated and tender, and owing to this twofold cause the tumor became imperceptible to the touch.

*Post mortem.* Sloughing and perforation of the lower portion of the ileum had taken place, and peritonitis had been set up. A large mass of scirrhus, readily broken up, and partly detached, and stained by the effused faeces, occupied the folds of the mesentery adjacent to the caecum. A small scirrhus nodule was found in the walls of the cystic duct, obstructing the passage. The gall bladder was distended with bile. No scirrhus existed elsewhere in the abdomen or in the chest.

The heart was reduced in volume; it weighed only six ounces. The muscular substance was of a dun-brown colour, firm to the touch, and in the granular stage of degeneration; the cavities and walls were relatively normal; the great vessels and the coronary arteries were reduced in size proportionately to the heart, but they were otherwise unaffected. The pericardium was universally and firmly attached to the heart, and corrugated; and the adventitious connecting medium was the seat, near the base of the heart, of several plates of osteoid formation, and of two so called cartilaginous laminae. The latter, examined microscopically, were found to consist of nucleated fibroid tissue, resembling non-striated muscular fibre after treatment with acetic acid. No cartilage corpuscles or lacunae existed.

Atrophy of the heart in this case was of later date than adhesion of the pericardium, as shown by the corrugated state of the latter, and was a consequence of the general failure of nutrition arising from the cancerous cachexy. As in the preceding case, the formation of osteoid and chondroid substance, in connexion with the products of antecedent pericarditis, was the result of failure of nutrition, and, in my opinion, coeval with atrophy of the heart.

CASE XLI.—*Leucocythæmia. Cirrhosis of the Liver, and Concentric Atrophy of the Heart.*

A gentleman, who had been a veterinary surgeon in the army, and had served in India for seven years, came under my notice professionally on the 18th September, 1862. He had been invalided, and after his return from India, had undergone a course of mercury for secondary syphilis. He was of very delicate frame, pale, nervous, and emaciated. The appetite was capricious, the bowels irregular, and sleep was obtained only by the free use of opium. The spleen was manifestly enlarged; but no other organic disease could be detected. He continued under my treatment for nine months. During that time he suffered from slight neuralgic pains in various parts of the body, from periostitis of the tibia and of the cranium, and he had an attack of testitis. The urine frequently exhibited a copious precipitate of lithate of ammonia. In the course of the winter, cachectic ulcers, apparently syphilitic, appeared upon the legs, and the feet became swollen. He was frequently unable to take food of any kind.

In the succeeding summer, under the advice of an eminent physician, he visited Barége and drank the waters; but, after six months' residence there, he returned in no degree improved. Whilst at Barége he suffered from continual diarrhœa, which he attributed to the use of the waters; the ulcers upon the legs, however, had healed.

I saw him again in September, 1864, when, in addition to anasarca, there was ascites; and on the 23rd November he became a private patient of the Mater Misericordiæ Hospital. He now began to suffer much from flatulent distention of the large intestine. A drop of blood, obtained from his finger by pricking with a needle, exhibited an excess of white corpuscles, which almost equalled the red corpuscles in number. The ascites increased rapidly, and his debility became extreme. The dejecta from the bowels, which were constipated, were of a light colour, and exceedingly fetid; there was no trace of jaundice.

He died, worn out, on the 21st December.

At the examination of the body, the left pleural cavity was

found nearly full of serum, and the lower portion of the left lung compressed. The right lung, which was emphysematous, presented five lobes instead of three. The heart was very small, not exceeding that of a child of five years; the right cavities were in all respects normal, proportionately to the size of the heart; but the left ventricle was in the condition of *concentric atrophy*, its walls being much thickened, and its cavity reduced in size. The substance of the heart and its valves was structurally sound.

There was a large collection of serum in the peritoneum, and the spleen was greatly enlarged. The liver was in an early stage of cirrhosis, and the gall bladder was distended with bile. The kidneys were somewhat enlarged, and exhibited a few patches of fatty degeneration. Attached by a fine pedicle to the posterior false ligaments of the bladder, were four, small, round bodies, of a yellow colour and smooth surface, consisting of a cortex, which was composed of small cells, and an enclosed plexus of capillary blood-vessels. For a more full report of this case, the *Proceedings of the Dublin Pathological Society*, New Series, Vol. ii., Part ii., 1864, may be consulted.

It is scarcely necessary to remark that the foregoing case illustrates atrophy of the heart produced by a general or constitutional cause of a non-malignant character; namely, the peculiar and probably specific blood-crisis, known as leucocythæmia.

The three forms of atrophy of the heart mentioned at the outset, will be found exemplified in the preceding cases.

I shall now proceed to the consideration of the special or *heteroplasic diseases* of the substance of the heart.

These may be discussed under the following heads, viz.:

1. Fatty disease.
2. Fibroid.
3. Cancerous.
4. Syphilitic.
5. Hydatid.
6. Tubercle.

In the discussion of these various subjects the above order will not be strictly observed.

*Fatty disease* of the heart, as conventionally interpreted, includes two conditions of that organ which differ widely in regard to their genesis, the anatomical lesion involved, the degree of impairment of function, and the benefit to be expected from treatment. These are, *fatty deposition or infiltration*; and *fatty degeneration*.

*Fatty deposition or growth* is simply an extension into the domain of disease of the physiological process of "growing fat." It consists in the absorption by cells of free fat from the blood. The connective tissue corpuscles of the sub-cutaneous areolar tissue, of that of the orbit, mesentery, etc., perform this process normally; and abnormally, the hepatic cells, and the corpuscles of the perimysium. It occurs more readily when there is excess of free oil in the blood.

From the mode of its production, fatty infiltration of the heart must be located externally to the sarcolemma of the muscular fibres; and in this situation it is exhibited in the form of fat cells of average size and configuration. In both these respects fatty infiltration differs anatomically from fatty metamorphosis, as will be presently shown.

In fatty infiltration, function is not abolished by destruction of tissue; it is only mechanically impeded by encumbrance and by partial absorption of the muscular fibre; and, finally, it is much less grave prospectively, and more amenable to treatment than fatty metamorphosis.\*

It may be conveniently divided into *superficial and interstitial* infiltration of the heart, as proposed by Quain. Of these, the former is normal, and the latter abnormal, anatomically considered.

*Fatty metamorphosis*,† or *degeneration* of cellular structures, consists, according to Rindfleisch, in a decomposition of the amalgam of albumen and fat, of which the cells are composed, the fat being set free, and in muscle collecting in the form of

\* *Rindfleisch Pathological Histology*, New Sydenham Society's edition, 1872, vol. i.

† Fatty metamorphosis, as employed by Rokitansky, means deposition of fat cells *between* the muscular fibres; not transformation of the sarcous elements into oil-clots, as implied in the text.



groups of oil dots in the interfibrillary protoplasm of the fibres. This aggregation may be observed especially around the nuclei, owing to the large size of the spaces formed here for their reception by divarication of the adjacent fibrillæ.\*

Hoppe-Seyler, Burdach, Kemmerich, Voit, and Ssubotin have shown that fat may be the product of the retrograde metamorphosis of albuminoid bodies.†

Fatty metamorphosis of cells occurs normally in the epithelium; for example, in that of the lactiferous and the sebaceous glands. But when it occurs pathologically it is due to a derangement of proportion between the means of nutrition and the structure to be nourished; and such disproportion may result either from a diminution of the *nutriens*, or an increase of the *nutriendum*.‡

Defect of pabulum may arise from a local or from a general cause. As examples of the former may be adduced, obstruction of the coronary arteries by atheromatous or calcareous change in their coats, or by fibrinous thrombosis or embolism,§ and the disturbance of circulation arising from the inflammatory process.

In parts recently inflamed there is an inadequate supply of pabulum, not only from the attendant disturbance of circulation, but likewise from mechanical arrest to the transit of nutrient material by massive proliferation of corpuscular elements. To the derangement caused by the defective supply of nutriment thence arising, may be added that resulting from increase of parenchyma by inflammatory products.

Fatty degeneration of the heart may likewise arise from general failure of nutrition (and this is its most frequent cause), whether from absolute want of the elements of a wholesome and sufficient dietary, as amongst the poor; from failure of tissue-assimilation; or from failure of circulation through athero-

\* *Antea*, p. 596.

† Doctor Macalister, in *Dublin Quarterly Journal*, August, 1870.

‡ Rindfleisch, *opus citat.*

§ It is generally held that the tissue-change which takes place in the heart, consecutive to thrombosis or embolism of the coronary arteries, being the result of an acute cause, is softening rather than fatty degeneration.

matous change of the arterial coats. To these changes gouty subjects of middle or advanced age are especially liable; hence the frequent association of gout, arterial atheroma, and fatty degeneration of the heart. Whether imperfect disintegration of albuminous substances in the liver is the cause of lithæmia, gout, and atheroma, as Dr. Murchison, with good reason, suspects to be the case,\* it is certain that atheromatous degeneration of the aorta and coronary arteries is the most frequent precursor of fatty degeneration of the heart.

Poisoning by phosphorus has been also an occasional cause of fatty degeneration of the heart and other viscera. Several examples of this kind may be found in the works of Orfila and Taylor; and a very striking example has been published by Dr. Habershon.† A woman, aged twenty-eight, took by mistake a quantity of rat poison, containing about five grains of phosphorus. She exhibited the usual symptoms of phosphorus-poisoning, and died on the fifth day after the occurrence. The liver, spleen, kidneys, and heart, as well as the voluntary muscles, were in a state of fatty degeneration. In the "Year-Book" of the New Sydenham Society for 1860, a case, published by Lewinsky, is referred to, in which death followed the taking of a large dose of phosphorus, and the liver was found to be "lardaceous."

In the volume of the same publication for 1863, another case of the same kind is quoted from the writings of Bucquoy, in which the brain was found to have undergone fatty degeneration.

When due to obstruction of the coronary artery, or to a general cause, fatty metamorphosis would be "primary;" and when dependent upon preceding inflammation, it would be "secondary," as classified by Quain. In this latter category I would include fatty transformation of the substance of the heart, supervening upon, or secondary to, hypertrophy.

Doctor Ponfick proposes the division of fatty degeneration of the heart into the "senile" or "plethoric;" and the "anæmic." The subjects of the former are aged and fat, the arteries are degenerate, and the costal and tracheal cartilages are calcified.

\* Croonian Lectures, *British Medical Journal*, 1874.

† *Medico-Chirurgical Transactions*, vol. l., 1867.

The fibres of the heart frequently exhibit pigmentary as well as fatty change.

The subjects of the second, or anæmic form, are young and anæmiated. There is slight fatty change of the internal and middle coats of the arteries, defect of red blood corpuscles and fibrin, and relative excess of white corpuscles. He adds, that in the former cases the fatty change is usually local, and in the latter it is general.\*

Quain notices the frequent association of recurrent hæmorrhage with fatty degeneration of the heart in young persons, and Virchow states that fatty degeneration of the heart is a common accompaniment of chlorosis.†

Having thus shown that there are two distinct forms of fatty disease of the heart, and defined briefly their genesis and anatomical differences, I shall proceed, in the next place, to give a succinct but necessarily imperfect history of the subject. For the materials of this sketch, prior to 1850, I am mainly indebted to the able and exhaustive memoir of Dr. Quain.‡

It is necessary, however, to mention at the outset, that owing to the disadvantage of a total want of acquaintance with, or an imperfect appreciation of, the distinction between fatty growth and fatty degeneration, under which the early writers on this subject were placed, a certain amount of confusion and obscurity was unavoidable in their description of symptoms and anatomical lesions. Hence, it will be necessary, in the classification of symptoms which have been assigned to fatty disease of the heart generally, to distinguish carefully between those which are proper to the one and to the other form, respectively.

Hippocrates remarks, that “persons who are naturally very fat are apt to die earlier than those who are slender;”§ and Celsus, that “whilst thin persons are feeble, fat persons are unenduring.”||

\* *London Medical Record*, February 19th, 1873, quoted from *Berliner Klinische Wochenschrift*, 1873.

† *Ibid.*, January 8th, 1873.

‡ *Medico-Chirurgical Transactions*, vol. xxxiii.

§ Works translated by Adams, *Aphorism* 44.

|| “Gracile corpus infirmum ; obesum hebes. est ;” *De Re Medica*, liber ii., sec. i.

Galen alludes merely to the inconvenience of corpulence, and to the necessity and the means for reducing it.\*

Harvey describes the heart of old Parr, who died in 1635, as laden with fat.†

Lancisi likewise alludes to fatty accumulation upon the heart.‡ And Morgagni gives some examples of this condition, of which the following deserves special notice on account of the symptom of fainting exhibited.

A very fat woman, aged seventy-five years, subject to fainting fits (*"quasi-deliquia"*) died suddenly. The mediastinum and the heart were loaded with fat, and death had occurred by rupture of the left ventricle on its posterior surface.§

Corvisart had never seen an example of fatty change of the fibres of the heart, but alludes to it as having been witnessed by others. He had, however, frequently met with examples of fatty accumulation upon the heart, and admits that this condition may be the cause of sudden death. ||

It would appear, therefore, that to Corvisart, although he had very little personal experience of the disease, is due the merit of having been the first to recognize the distinction between the two forms of fatty change of the heart.

Laennec, whilst describing minutely the different forms of "softening of the heart,"¶ does not even allude to accumulation of fat upon it, or to fatty transformation of its substance.

Doctor A. Duncan junior, in 1816, published the case of a female, aged twenty-two years, who died rather suddenly after severe and protracted pain in the region of the heart. The pericardium was found universally adherent, and the substance of the heart, to the depth of two-thirds of its thickness, replaced by fat.\*\*

In 1818 Cheyne published his well known case, which merits special notice in a historic summary of this subject, because cer-

\* *Methodus Medendi*, liber xiv., c. xv.

† Works translated by Willis, Sydenham Society, p. 590.

‡ *De Motu Cordis, Romæ*, 1728, p. 55.

§ *De Sedibus et Causis Morborum*, epistola xxvii., observation 2.

|| *The Diseases of the Heart*, Hebb's translation, pp. 166 and 168.

¶ *Auscultation Médiate*, tom. ii., p. 533.

\*\* *Edinburgh Medical and Surgical Journal*, 1816.



tain specific symptoms are here for the first time noticed in connexion with a fatty condition of the heart.

A full, stout, and florid man, aged sixty years, addicted to the pleasures of the table, gouty, and subject to occasional attacks of catarrh, exhibited for the last few years of his life oedema about the ankles in the evening, and occasional intermittence of the pulse.

After a long and fatiguing walk he had fluttering and palpitation of the heart, followed by a "fit," which was believed to be apoplectic, in which he lost consciousness; and the pulse became irregular and unequal. For this, which was followed by a sense of fulness and pain in the head, he was bled to nearly twenty-eight ounces within a period of little more than an hour; and shortly afterwards he had fifteen leeches applied to the head.

Under this treatment he recovered, save as to the pulse, which continued irregular and unequal. The feet and legs soon afterwards became swollen, and he had a slight attack of articular gout. He survived the fit about two months, and for the last few days of his life the respiration was irregular; it would cease for a-quarter of a minute, then be feebly resumed, increase gradually in rate and energy, and then gradually subside till it was again suspended. In Cheyne's words: "For several days his breathing was irregular; it would entirely cease for a-quarter of a minute; then it would become perceptible, though very slow; then, by degrees, it became heaving and quick; and then it would gradually cease again. This revolution in the state of the breathing occupied about a minute, during which there were about thirty acts of respiration." Shortly before death he became hemiplegic (right) and aphasic. The brain was found congested over the left hemisphere. Two ounces of serum had collected in the pericardium. The heart was three times its natural size, and the right ventricle was entirely converted into fat in its lower part, and very thin superiorly. The left ventricle was greatly dilated and converted into fat, save its internal reticular part and papillary muscles.

The valves were all healthy, but "the aorta was studded with steatomatous and earthy concretions."\* The remarkable change

\* *Dublin Hospital Reports*, vol. ii., p. 216.

in the rhythm of respiration, of which this case presents the first example on record, was due, not to the condition of the heart, but to that of the aorta, as will appear in the sequel.

Doctor Gratiloup, of Bourdeaux, gives the case of a curate of that town, who died suddenly whilst in bed. On examination of the body, the heart was found greatly loaded with fat, and death had resulted from rupture of the right auricle.\*

Mr. Adams is the next writer who illustrated this subject by cases and remarks, which, studied even in the light of present knowledge, will be admitted by all candid readers to possess extraordinary merit for originality and accuracy.†

All Mr. Adams' cases, like those hitherto noticed, are examples of fatty growth.‡ He describes accumulated fat on the heart, a thin layer of muscle only remaining internally, and expresses the opinion that death in such cases may be sudden, and preceded by the usual symptoms of apoplexy. The two following cases are examples :

A gentleman sixty-eight years of age, had, in the course of seven years, no less than twenty pseudo-apoplectic fits, in which the breathing was stertorous and irregular, and the pulse slow ; but no paralysis had succeeded. He was usually heavy and lethargic, and the memory failed at the approach of these fits. At the autopsy, both ventricles were found laden with fat, muscular tissue to the depth of a line only remaining on the internal surface.

A physician, aged sixty-eight years, who had had fainting fits with momentary loss of consciousness several times in the course of the last ten years of his life, was suddenly seized with pain in the chest, vertigo, and dyspnœa, on making an unusual effort with his arms lifted above his head. For six weeks, during which

\* *Archives Générales de Médecine*, 1822.

† *Dublin Hospital Reports*, vol. iv., 1827.

‡ Doctor Ormerod has stated incorrectly, that the cases published in Dublin, prior to 1849, were examples of fatty degeneration, which he designates as "the Dublin form" of fatty heart. I have carefully looked into all the cases recorded in Dublin, and, with the exception of those published by Drs. Townsend and Stokes, I have failed to discover amongst them, previous to 1849, any examples of *fatty degeneration*, entitled to be so regarded from the description of them given by their authors. Of *fatty growth*, the cases published in Dublin before that date are numerous ; and since then, both forms have been amply illustrated by the Dublin School.

he lived subsequently, there was no pulse to be felt in any of the arteries of his body, and no cardiac impulse beyond slight fluttering. He died worn out, and in a state of coma. The heart was found large, and loaded with fat; the aortic valves were completely calcified, and the first inch of the coronary arteries was calcified and entirely closed.

Doctor Townsend published\* two cases of sudden death by rupture of the left ventricle in aged subjects. The walls of the left ventricle were "pale, soft, and flabby," and, in the seat of rupture, in an advanced state of fatty degeneration. At the base of the heart, and in the sulci, there was a thick layer of fat; and the walls of the right ventricle, which was more than half an inch thick, was composed of fat, with a layer of muscle, of only the thickness of paper, on its internal surface. In both these cases the coronary arteries were extensively calcified and partially closed. To this, as the cause of imperfect nutrition of the heart, Dr. Townsend attributes the fatty change which the organ had undergone. He likewise adverts to the absence of ulceration in the seat of rupture, and dwells upon the adequacy of fatty deposition and softening of the walls of the heart, to account for this accident.

Doctor C. J. B. Williams alludes to the subject of fatty softening of the heart.†

Professor R. W. Smith was the next contributor to the elucidation of this subject. He published two remarkable cases, in which death occurred so soon after admittance to hospital, that no opportunity was presented for noting symptoms. In both cases the heart was laden with adeps, *and free oil was found in the blood*. He expressed the opinion, since confirmed by the highest authorities in pathology, that the free oil found in the blood in these cases was of "constitutional origin," and due to imperfect assimilation, being an excess of the fat normally found in the blood.‡

Doctor Latham, in 1839-40, recorded four cases, all of which were good examples of fatty accumulation and fatty degenera-

\* *Dublin Journal of Medical and Chemical Science*, 1832, vol. i., p. 165.

† *Pathology and Diagnosis of Diseases of the Chest*, third edition, 1835, p. 191.

‡ *Dublin Journal*, vol. ix., July 1st, 1836.

tion combined. All four patients were males, and over sixty years of age, and two at least of the four were gouty. Two of the patients were subject to fainting fits; and in these, circumscribed aneurism of the left ventricle existed in conjunction with a fatty state of the heart, but rupture of the heart had not taken place, and death occurred by syncope. In the two remaining cases, rupture of the *septum ventriculorum* was the immediate cause of death; one of the patients surviving the occurrence of this accident eighteen hours, and the other three days, as judged by the final symptoms. Both these patients had had one or more attacks, resembling *angina pectoris*, in their last illness.

Doctor Latham held that a fatty state of the heart, however advanced, did not admit clinically of positive diagnosis; its symptoms and signs warranted only "a probable conjecture;" and even these were due, not to the structural alteration in the heart, but to the associated condition of dilatation which usually supervenes at an advanced stage of the affection.\*

Four years later, Mr. Carmichael published an example of both forms of fatty change of the heart combined in the same individual. A clergyman, aged upwards of sixty years, temperate, but for a long time subject to palpitation and fainting fits, died suddenly, with symptoms of apoplexy, whilst at a wedding breakfast.

On the day succeeding that on which he died, the superficial veins of the forehead became turgid with air, and crepitant. The heart was covered with fat; the walls of the right ventricle, reduced to two lines in thickness, were almost entirely composed of fat, the remaining muscular tissue being greasy and readily broken down. The left cavities presented a similar condition, but in a less degree.†

In the same session (1840-41), Mr. Fleming communicated to the Pathological Society the particulars of a case of a similar character, but more valuable, because of the history of the patient's last illness, extending over several months, which he possessed. A gentleman, aged forty-five years, was rather sud-

\* *Lectures*, vol. ii., lecture xxvi.

† *Proceedings of the Pathological Society of Dublin*, Session 1840-41.



denly seized with nocturnal dyspnoea and palpitation, and shortly afterwards with loss of consciousness and hemiplegia. This latter attack lasted five hours, and during its continuance the pulse was nearly imperceptible. Under the use of a mustard foot-bath, a sinapism to the nape, and the administration of camphor and ammonia, recovery took place. There were four similar attacks between March and November, all of which were attended with jaundice, and were successfully treated in the same way. The heart's action was strong, and accompanied with *bruit de soufflet*; the radial pulse was small and feeble, and occasionally imperceptible. The application of even a single leech caused alarming weakness, and there was extreme intolerance of opium; death was caused by another disease. The mitral valve was thick and incompetent, the left auriculo-ventricular opening very large, and there was excentric hypertrophy of the left ventricle. The lungs were congested, partially emphysematous, and contained some apoplectic nodules. The state of the substance of the heart was not mentioned; but, from the symptoms, I think it may be regarded as fatty.

Doctor Stokes recorded the particulars of a case of sudden death by syncope in a man aged fifty years, with haggard expression of face, weak, but regular pulse, and feeble action of the heart, the sounds being faint, but distinct. The right ventricle was found loaded with fat, the left ventricle soft and flabby, and the valves all sound.\*

In 1846 the following case was likewise noted by the same observer. A man aged fifty years was admitted into the Meath Hospital. He was very weak. The pulse varied from 35 to 40, but was regular. A systolic murmur existed in the ascending aorta, loudest at the right second costal cartilage. Death was sudden. The heart was soft and flabby, and covered by a thick layer of fat. The aortic valves were diseased, and the orifice narrowed. The aorta was atheromatous.†

Vicq-d'Azyr,‡ and Partridge§, in 1847, published examples

\* *Proceedings of Pathological Society*, November 25th, 1843; *Dublin Journal*, vol. i., new series, p. 491.

† *The Diseases of the Heart and Aorta*, p. 312.

‡ *Œuvres par Moreau*, tom. v., p. 365.

§ *Medical Gazette*, 1847, p. 944.

of fatty degeneration of the voluntary muscles from paralysis; and in the same year Mr. Paget's important lectures, illustrative of the same subject, appeared.\*

Doctor Bellingham exhibited before the Surgical Society of Dublin an example of fatty accumulation on the heart, especially on the right ventricle, with double aneurism of the arch of the aorta, taken from the body of a man aged twenty-seven, who died suddenly in a state of syncope. During life this man had exhibited no symptoms indicating a fatty condition of the heart.†

In the same year, and before the same Society, Dr. Henry Kennedy read a valuable paper on the subject of fatty disease of the heart; but chiefly in advocacy of the treatment of that disease by bodily exercise. He gave the particulars of two cases, which seemed to be examples of the compound form of the affection; that is, of fatty accumulation and fatty degeneration combined. In both cases the patients were of advanced age, and subject to fainting fits without loss of consciousness.‡

In the discussion of Dr. Kennedy's paper, Professor Harrison, while dissenting from the plan of treatment recommended, exemplified the danger of exercise in fatty disease of the heart, by reference to the case of an old gentleman who, whilst making a journey in his carriage, suddenly died in consequence of a jolt of the vehicle. The heart was found laden with fat, and its substance in a state of fatty degeneration.

Doctor Latham Ormerod in this year (1849) formally recognized two distinct varieties of fatty change in the heart; viz., fatty accumulation on the heart in fat persons, and fatty degeneration of its substance.§ The latter form was that which was especially treated of in his memoir. This he describes as exhibited in fawn-coloured patches on the inner or outer surface of the heart; and, microscopically examined, these patches are seen to exhibit the following changes. The fibres immediately adjoining show interruption in the continuity of the transverse striæ, which now appear as transverse lines of dark dots; more

\* *London Medical Gazette*, 1847, lectures v. and vi.

† *Dublin Medical Press*, vol. xxi., April, 1849.

‡ *Ibid.*, vol. xxii., December, 1849.

§ *London Medical Gazette*, vol. ix., p. 739.

internally the intervals between the dots are longer, and the latter assume a linear arrangement in the length of the fibre; and still nearer the centre they are scattered irregularly, constituting the "granular condition," or granular stage. Towards the centre of these patches oil dots appear at distant intervals, and finally they take the place of the dark granules within the sarcolemma; this is the "fatty stage." The condition just described he declares he has never met with in the auricles. He denies chemical "conversion" into fat; but admits a "substitution" of fatty for the disintegrated muscle-particles in the process of nutrition.

In 1853, Paget's great work appeared, in which, amongst many subjects, that of fatty changes in the heart is fully considered.\*

In 1854, Dr. Stokes' valuable clinical treatise† was published; in it fatty change of the heart in its two forms is amply discussed and illustrated. Since that date the contributions to the pathology and the clinical history of fatty disease of the heart have been very numerous, and the affection may be considered to have taken a recognized position in nosology.

The two forms of fatty change of the heart are usually, but not always associated, and the apparently opposite circumstances under which they are met with may serve to explain both the rule and the exceptions.

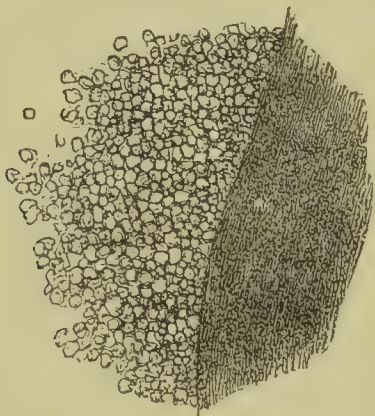
Fatty growth upon the heart is most frequently associated with general obesity, and occurs at that period of life, and in that class of society, in which deposition of fat is of ordinary occurrence in the natural or physiological order. But beyond a certain limit, obesity is not compatible with the active nutrition, or the functional vigour of muscle, because of the impediment to muscular exercise, and the consequent impairment of muscular nutrition, which it involves. In many instances, also, it is associated with the gouty diathesis and the attendant atheromatous change in the arteries, by which the nutrition of tissue is directly interfered with through an inadequate supply of blood.

\* *Lectures on Surgical Pathology*, vol. i., p. 93, *et sequent.* In some valuable lectures published in the *London Medical Gazette* several years previously, Mr. Paget had fully recognized the distinction between fatty growth and fatty metamorphosis.

† *The Diseases of the Heart and Aorta.*

In many cases, likewise, by the mere weight of incumbent fat, muscle undergoes absorption and atrophy; and where, as most frequently happens, these concomitant changes are met with in the heart, even the residue of muscular substance is unsound. Nevertheless the portion of muscular fibre as yet unaffected by the pressure of the adipose mass, may, if uninfluenced by the other causes of malnutrition previously mentioned, be actually found in a state of textural integrity. This condition is, however, strictly exceptional, and a good illustration of it is presented in the subjoined engraving (Fig. XXXIV.) from the heart

FIG. XXXIV.



Fat cells and muscular fibre; magnified 24 diameters. Mr. B.

of Mr. B. (*vide* Aneurism). It will be observed that whilst the accumulated fat has caused, by its pressure, all but complete absorption of the walls of the right ventricle, or at least, has replaced the muscular substance of the walls nearly to their entire depth, the thin muscular layer which remains is histologically sound. It is further noticeable that the line of union between the adipose substance and the muscular structure of the heart is quite abrupt, and that no fatty infiltration of the latter has taken place. In Fig. XXXV. the adipose tissue from the surface of the same heart is represented still more highly magnified; and in the next engraving (Fig. XXXVI.) the unaltered muscular fibre, equally magnified, is represented.



FIG. XXXV.

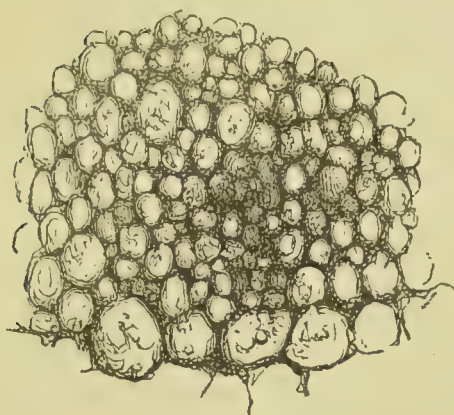
Fat cells ;  $\times$  210 diameters. Mr. B.

FIG. XXXVI.

Unaltered muscular fibre ;  $\times$  210 diameters. Mr. B.

In this case there were present none of the ordinary symptoms of fatty disease of the heart, except the comparatively insignificant one of weak pulse; and no physical sign indicative of that condition, with the single exception of a feeble first sound. Death took place by rupture of a large aneurism of the aorta into the pericardium.

Judging from the experience afforded by this single example of fatty growth upon the heart, causing absorption in great part of its proper structure, but unattended with fatty infiltration or fatty degeneration of that portion of the muscular wall which remained (and it is the only one of the kind which has come

under my notice), I would say that simple accumulation of fat upon the heart is, *per se*, unannounced by evidence, either general or special, sufficient to warrant a positive diagnosis. In all the examples of sudden death by simple syncope, or by rupture of the heart, associated with epicardial growth of fat, which I have met with or seen recorded, there was reason to conclude, where a positive statement to that effect was not made, that the muscular substance of the heart had undergone, in a greater or less degree, the change of fatty degeneration. Thus, it will be found that the muscular structure of the organ was represented as being fawn-coloured, easily torn, or soft and flabby. Any one of these statements would justify the suspicion of fatty change of the muscular fibres.

Fatty growth upon the heart is usually traceable to causes identical with those which give rise to general obesity; especially to sedentary habits, and indulgence in spirituous liquors.

True *fatty degeneration*, or transformation of the muscular fibres of the heart, is a much more serious affection, and differs from the preceding genetically as well as histologically.

*The pathology* of this form of the affection, quite irrespectively of the estimate which may be formed by individuals of the value of pathological doctrines generally, is of undoubted importance in regard to its probable issue and treatment.

Doctor Latham Ormerod maintained that fatty degeneration is the product of a veritable perversion of nutrition, attended with sub-oxidation of the food and of the tissues, and resulting in the deposition of morbid fat or olein.\* He denied the identity of adipocire with the product of fatty degeneration.

This is likewise the opinion of M. Ch. Robin, who designates the change as "fatty substitution."<sup>†</sup>

Mr. Paget, on the contrary, is of opinion that it results from a *defect* of nutrition, and consequent chemical changes of tissue of an atrophic character.<sup>‡</sup>

Rindfleisch, as already stated, holds the same opinion.

Doctor Quain regards the change as physical or chemical, and

\* *St. Bartholomew's Hospital Reports*, vol. iv., 1865.

† *Chimie Anatomique*; see Flint, *Diseases of the Heart*, second edition, 1870, p. 97.

‡ *Lectures on Surgical Pathology*, 1853, vol. i., p. 93.

analogous to the decay of dead structures outside the body. It is, therefore, in his opinion, closely allied to, if not identical with, adipocirous transformation.\*

I believe that fatty degeneration of muscle is a veritable necrobiosis, differing only in degree from the changes which take place when the controlling influence of tissue-vitality has been entirely withdrawn. In the former case, the process of tissue-death is slow and progressive, under the modifying influence of a feeble and inadequate nutrition. In the latter, nutrition being entirely and finally suspended, it is rapid and complete.

The circumstances which so impair the nutrition of the heart, as to induce fatty transformation of its substance, may be local or general. In the former case the transit of nutrient material is mechanically obstructed, as by calcification or occlusion of the coronary arteries, or some of their branches. Thus a larger or smaller district of muscle, according to the extent of arterial disease, or the size of the vessel affected, may be partially or wholly deprived of pabulum, and, under the influence of vital or physical agency, assume new forms according to its chemical affinities.

Most authorities are agreed that a similar series of changes may be induced by antecedent inflammation of the substance of the heart. Hence the gravity of myocarditis. Bouillaud, Hope, Virchow, and Rindfleisch advocate this view.†

Virchow maintains that dilatation of the heart, as implying antecedent irritation or sub-inflammation of its substance, has a similar effect.

The constitutional causes of fatty degeneration of the heart are such as interfere with general nutrition. Amongst these may be mentioned, long continued inadequate supply of food, especially if associated with severe physical labour, recurrent hæmorrhage, and tubercular or cancerous marasmus.

Drs. Ormerod and Quain call special attention to the frequent association of fatty degeneration of the heart with habitual loss of blood. Of this I have noted several examples.

\* *Medico-Chirurgical Transactions*, vol. xxxiii., 1850.

† Wagner, on the authority of Reynaud, states that fatty degeneration followed pericarditis in seventeen out of thirty-five cases in his practice. (See Flint, *Diseases of the Heart*, 1870, p. 98).

The above mentioned general causes, having reference to insufficient nutriment and excessive labour of body, apply especially to the poor and working classes. On the other hand, those of a local character, involving atheromatous or calcareous transformation of the coronary arteries, and inflammatory softening of the heart, are found to operate in about equal proportion upon the rich and gouty, and the poor and ill fed members of the community.

Thus, whilst excessive deposit of fat upon the heart is the almost exclusive appanage of the rich, fatty degeneration of its substance without excessive superficial deposit, is apportioned about equally between the rich and the poor. When it is remembered that, in the great majority of examples of fatty growth upon the heart, fatty degeneration of its muscular structure likewise exists, it will be conceded that the wealthy classes of society enjoy an unenviable preeminence in regard to liability to fatty disease of the heart.

This conclusion is not quite in accord with that at which Quain has arrived. Of forty-eight cases of both forms of fatty change of the heart, respecting which he has noted the class of society to which the patient had belonged, sixteen were of the upper, fourteen of the middle, and eighteen of the lower class.

The process of fatty transformation of the heart may be conveniently divided into three stages, as determined by microscopical inspection: namely, the stage of *granular transformation* of the fibres, that of their *substitution by oil drops*, and the stage of their *obsolescence*. Dr. Ormerod maintains that the granular is distinct from the fatty change, and involves no increase of oil in the muscular substance. In this opinion I cannot agree with him, and I find myself rather in accord upon this point with Drs. Quekett and Quain, who regard the granular transformation of the muscular fibres as representing the first stage of their conversion into fat.\*

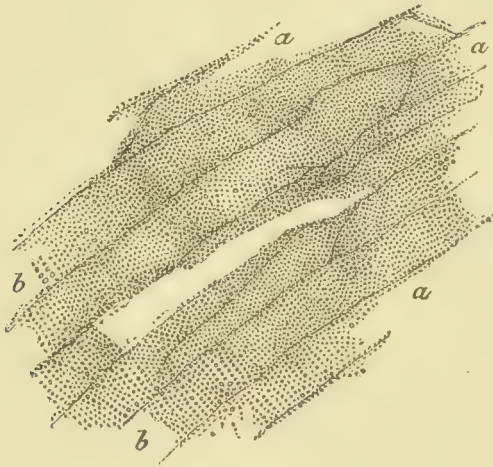
In this stage, the muscular fibres are well defined in outline, and present transverse striation with the usual distinctness over a greater or less extent of the field of vision, but inversely as the progress in histolytic change actually made. Over a variable portion of the field, likewise, and directly as the advance of fatty

\* *Lectures on Histology*, p. 196, and *loco citat.*



change, the transverse markings of the fibres are interrupted, and represented by lines of dark molecules at right angles with the axis of the fibres. In certain parts of the same section, and indicating a more advanced degree of change, these transverse lines of dark molecules are replaced by longitudinal rows of similar bodies; this is apparently due to the twofold cause of derangement of mutual apposition of the fibres, and conversion of entire fibrils or strings of sarcous elements into minute oil dots. The condition indicated, under the various transitional changes just mentioned, is represented in a preceding engraving. Fig. XXXI., p. 553, shows the granules at a very early period, when the parallelism of the transverse series is scarcely deranged; and Fig. XXXVII. (Mary A.) subjoined, shows the granular transformation in a still more advanced stage.

FIG. XXXVII.



Granular degeneration.

(b) Early stage; disintegration of transverse striæ into granules.

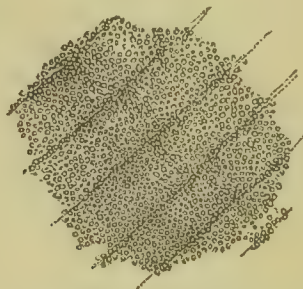
(a) A more advanced stage; longitudinal arrangement of granules.

× 222 D. Case 61 (Mary A.).

In the further progress of change, the minute dark granules coalesce and form larger spherules or oil dots, distinguishable by a luminous centre and a dark outline. These differ considerably in size, and are disposed at unequal intervals in the length of the fibre. They are usually crowded around the nucleus, as noticed by Paget and Rindfleisch. The latter assumes, but in my opinion incorrectly, that the appearance is due to the diffusion

of oil amongst the fibrils, and its accumulation in the interstices formed around the nucleus by their divarication. It is not difficult to satisfy one's self that the molecules of oil, when thus aggregated, are still within the limits of the fibrillary walls, and though grouped around the nucleus, preserve the linear arrangement of the fibrils. This crowding of the oil dots within their respective fibrils, I incline to attribute rather to the vital or nutritive attraction of the nucleus than to a simple mechanical agency. In a more advanced state of transformation, what may be designated the third stage, or that of obsolescence after Ormerod, the fibrils would seem to have undergone solution, and the contained oil to have become diffused, forming large oil drops within the sarcolemma. All appearance of definite structure in the fibre is now utterly effaced; large portions of it present a perfect blank, and in these situations, as determined by its still well defined outline, it is reduced in diameter, whilst it is enlarged or bulged out in those portions where the oil drops have congregated. It rarely happens that this advanced degree of change has extended beyond a very limited area, because its further progress is usually interrupted by death, arising from rupture of the heart where it has been attained, or from syncope without apparent disintegration of structure. The accompanying engravings (Figs. XXXVIII. and XXXIX., John McD.) show the several anatomical peculiarities of the second and third stages just described. Where fatty degeneration is due to obstruction of minute branches of the coronary arteries, its area is often very limited. It not unfrequently also ap-

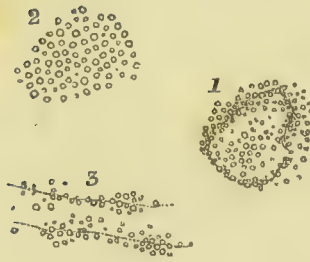
FIG. XXXVIII.



John McD.  $\times 222$  diameters.

Fatty degeneration (early stage). See Case 57.

FIG. XXXIX.



John McD.

- (1) Transverse section of fibre ; sarcolemma visible and partially collapsed, with numerous escaped oil dots.
- (2) Transverse section of fibre ; sarcolemma not visible.
- (3) A fragment of a fibre seen in length, showing partial obsolescence. The oil drops are collected in groups in the situation of the nuclei ; but most of them have escaped from the sarcolemma. See Case 57.

pears upon the inner or outer surface of the heart, in the form of light fawn-coloured spots, varying in size from the diameter of a pin's head to that of a split pea. This appearance is most frequently observed on the papillary muscles and fleshy columns, and is most probably due to antecedent endocarditis or pericarditis, as the neighbouring serous surface is generally opaque, and the endothelium thickened. In either case, these limited areas present, as it were in concentric circles, the different stages of fatty transformation above described ; the outer zone exhibiting the earliest condition of granular change, and the centre that of complete abolition of structure, as noticed by Ormerod. Whilst, in the majority of cases, superficial accumulation of fat upon the heart coexists with fatty degeneration of its substance, interstitial or interfibrous deposition of fat is very rarely met with in conjunction with fatty degeneration ; unless, as remarked by Sir J. Paget, the oil which has escaped by disintegration of the sarcolemma, be so regarded.

The *period of life* at which fatty degeneration most frequently occurs, though advanced, is not absolutely limited. From Tables V., VI., and VII.,\* comprising a total of ninety-five cases of fatty degeneration of the heart collected from three sources ; namely, the Transactions of the Pathological Society of London since

\* *Vide* p. 648, *et seq.*

1850, those of the Pathological Society of Dublin subsequent to the same date,\* and my own private records, I find the following results in regard to the ages of the subjects of this affection, given in decennial periods.

				<i>Cases.</i>	
Under 20 years	{	London Pathological Society	...	6	} = 9
		Dublin Pathological Society	...	2	
		The Author's returns	...	1	
20 to 30 years	{	London Pathological Society	...	6	} = 9
		Dublin Pathological Society	...	2	
		The Author's returns	...	1	
30 to 40 years	{	London Pathological Society	...	5	} = 6
		Dublin Pathological Society	...	0	
		The Author's returns	...	1	
40 to 50 years	{	London Pathological Society	...	10	} = 22
		Dublin Pathological Society	...	2	
		The Author's returns	...	10	
50 to 60 years	{	London Pathological Society	...	4	} = 8
		Dublin Pathological Society	...	2	
		The Author's returns	...	2	
60 to 70 years	{	London Pathological Society	...	13	} = 27
		Dublin Pathological Society	...	9	
		The Author's returns	...	5	
70 to 80 years	{	London Pathological Society	...	6	} = 7
		Dublin Pathological Society	...	0	
		The Author's returns	...	1	

From these combined returns, it would appear that fatty degeneration of the heart is most prevalent between the ages of sixty and seventy years; and next, between forty and fifty. The youngest patient was five, and the oldest seventy-nine years. My own returns show it to be most common from forty to fifty years, and *twice* more frequent within that decennial period than

\* As Doctor Quain's statistics (*Medico-Chirurgical Transactions*, vol. xxxiii.) were compiled in 1850, and contain in abstract all the cases previously published, I deemed it most convenient, and possibly most conducive to the interests of medical science, to take this date as my starting point, with the view of presenting one unbroken series up to the present time. With that object in view, I have, as far as was consistent with fulness of detail, assimilated my own returns, and those of the Dublin Pathological Society, to the Tables of Drs. Ormerod and Quain.



between the ages of sixty and seventy. I believe, indeed, that the disease under consideration belongs especially to the middle period of life, from forty to sixty years; 40·5 is the average given by Ormerod.

With regard to *sex*, Bizot maintains that fatty degeneration of the heart is more common in women than in men.\* Herein he is undoubtedly in error, as the following summary will show. Of a total of 55 cases collected from the London Pathological Society's Transactions, 35 were males and 20 females. A total of 18, obtained from the Transactions of the Dublin Pathological Society, furnishes 12 males and 6 females; and my own 22 cases yield 17 males and 5 females. Thus, out of a total of 95 cases obtained from these three sources, 64 were males, and only 31, *or less than one-half*, were females. Of Ormerod's 25 cases, 18 were males and 7 females; and Quain found the male subjects of the disease more numerous than the females, by 4 to 1.

The general condition of the patients may vary considerably; 9 of Ormerod's were fat, 6 were thin, and 2 were pale. Amongst his 25 cases there were 10 inebriates.

The *duration of illness*, from the date of the earliest symptoms connected with the disease to its fatal termination, varied in Quain's cases from a *minimum* of two to a *maximum* of 16 years. In Table V. (London Pathological Society), it varied from 4 days to 8 years. In Table VI. (Dublin Pathological Society), the extremes were 8 months, and 40 years; and in my cases, 3 weeks, and 16 years. Ormerod traced the origin of the disease to hæmorrhage in 4 cases, to phthisis in 3, and to anasarca in 10 cases. It is manifestly impossible to fix, even approximately, the date of commencement of structural alteration of the heart. It is only when this has made some, and even considerable progress, that it is declared by symptoms sufficiently pronounced to arrest the patient's attention. Thus, in a mere historic sketch of the case, the duration of ill health is usually under-estimated. Patients have repeatedly informed me that the first intimation which they had of anything serious being amiss with them, consisted in the sudden accession of violent pain in

\* *Memoires de la Société d'Observation*, tom. i., p. 351.

the region of the heart or epigastrium, with palpitation and dyspnœa on making some unusual physical effort, or on hearing some startling news. After this event they have never been again in their accustomed health (see Cases 23 and 51). In such cases a heart already far advanced in disease, but hitherto competent to discharge the routine duties of a quiet existence, has been surprised and overwhelmed by the sudden imposition of an extraordinary task, and never again been able to recover its dynamic equilibrium.

Again, in a case where structural disease of the heart had never been suspected, so robust had the health apparently been, the first announcement of its existence may consist in the rapid sinking of the patient, under the shock of an otherwise trivial acute disease. In this way, a mild attack of influenza, of bronchitis, of rheumatic or essential fever, may prove unexpectedly and rapidly fatal.

Mr. Paget truly remarks: "They who labour under it (fatty degeneration of the heart) may be fit for all the ordinary events of a calm and quiet life, but they are unable to resist the storm of a sickness, an accident, or an operation."\*

Where a person, the subject of fatty degeneration of the heart, is attacked with acute disease, the pulse becomes very rapid, as truly remarked by Dr. H. Kennedy. Hence the evil augury of very rapid pulse in febrile disease.

I have already intimated that fatty degeneration of the heart is not unfrequently secondary to hypertrophy, whether the latter affection be the result of valvular lesion, atheroma of the arteries, or Bright's disease.

Doctor T. K. Chambers found the heart enlarged in 29 out of 49 cases of fatty degeneration. And in 12 out of 35 cases of hypertrophy of the heart, secondary to renal disease, Wagner discovered fatty degeneration of its substance.† In 31 out of the 95 cases summarised in my Tables (V. VI. and VII.‡), hypertrophy existed in conjunction with fatty degeneration of the heart; that is, in 13 out of 55 in Table V.; in 4 out of 18 in

\* *Lectures on Surgical Pathology*, 1853, vol. i., p. 129.

† Flint, *Opus citat.*

‡ *Vide* p. 648, *et seq.*

Table VI.; and in 14 out of 22 in Table VII. This is, no doubt, most commonly the immediate cause of the fatal symptoms, when death proceeds from the heart, in cases of hypertrophy from renal or valvular disease, or from atheroma of the aorta. Where failure of the heart in a gouty subject, as indicated by irregular action and palpitation, quickly followed by paroxysmal dyspnœa and dropsical effusion, becomes the signal of a "break up" of the constitution, as not unfrequently happens, fatty degeneration and dilatation of the heart will be found the last links in the chain, of which atheromatous change of the aorta, hypertrophy of the heart, and granular degeneration of the kidneys constitute the preceding links, and inversely as the order in which they have been just mentioned.

That fatty degeneration of the heart may arise, as has been alleged, from the tuberculous or cancerous cachexia; from pyæmia or septicæmia; or as a result of any of the essential fevers,\* irrespectively of inflammatory complications of the heart, is, I think, more than doubtful. I believe, however, that slow poisoning by phosphorus or arsenic may give rise to it, although I have not myself met with an instance of the kind.

The *symptoms* indicative of a fatty condition of the heart are individually of little value; but combined in groups, as will be presently shown, they assume an affirmative significance.

I have already intimated that mere fatty growth upon the heart, without degeneration of its muscular substance, is not announced by any special symptoms or signs, save in so far as it may impede the motions of the heart mechanically, or cause thinning of its walls by continuous pressure upon them.

The symptoms regarded as proper to fatty degeneration of the heart have reference to the organs of the circulation, the brain, and the lungs; and, in a subordinate degree, also, the kidneys and the liver.

There is, in all cases, evidence of partial failure of the circulation, under the form of weak, irregular, intermittent, or very slow action of the heart and radial pulse; precordial oppression

\* Doctor Murchison, in the first edition of his great work, *A Treatise on Continued Fevers*, 1862, declared typhous softening to be identical with fatty degeneration of the heart.

or pain of an intermittent character, the latter frequently extending down the left arm as far as the elbow; palpitation on making any unusual effort, physical or mental; inability to resist the operation of heat or cold, or preserve the thermal equilibrium under extremes of temperature; pallor of surface; readiness to perspire; and recurrent syncope.

These symptoms of a feeble circulation were present in greater or less number in 42 out of the 95 cases represented in these Tables; that is, in 15 of Table V.; 12 of Table VI.; and 16 of Table VII. They were exhibited in 35 out of 108 cases given by Ormerod and Quain. They are rarely exhibited in the aggregate in any case of fatty transformation of the heart; nor when present as ordinary features, are they constant in any given case. The most frequently present and the most constant, are weakness or inability to endure fatigue, feeble pulse, palpitation and precordial oppression, accompanied by dizziness and a tendency to syncope after an effort of any kind, and want of the power of thermal adjustment.

The symptoms enumerated by Beau, Reynaud, and, more recently, by Seitz,\* as indicative of "strained heart" (*cœur forcé*, Beau); viz., breathlessness and palpitation, accompanied by a feeling of weight and oppression at the heart after trivial exertion; and, after a more severe effort, pain and a sense of constriction in the region of the heart, followed by chill, cough, and hæmoptysis, and occasionally by syncope, deserve special notice. The symptoms assume a more urgent character in the progress of the case, and are accompanied by a feeling of impending suffocation. Venous congestion, œdema, passive engorgement of the liver, and effusion into the serous cavities, follow in succession. The pulse is irregular and feeble. Precordial dulness may be either normal or extended; but the impulse of the heart is weak, and its sounds are dull and ill pronounced. Usually there is no cardiac murmur; but occasionally a faint murmur is heard with the first sound, or an indistinct *frottement*. Death may take place suddenly, or by a slow process of asphyxia.

\* *Deutsches Archiv f. Klinische Medicin*, vols. xi. et xii.



On examination of the body after death, no evidence of organic disease of the heart, with the exception of a few muscular fibres, "here and there," in process of fatty degeneration, can be discovered.

The history above given, and the symptoms enumerated, are certainly those of fatty disease of the heart; and in every case in which they have been presented to me, the heart, when examined after death, has been found in a more or less advanced stage of fatty change.

Irregularity of the pulse is somewhat more common than intermittence, and both are inconstant, and associated with temporary exhaustion. Irregularity of pulse was present in 5, and intermittence in only 1 out of 95 cases. The triple combination of pain in the region of the heart, dyspnoea, and syncope, constituting angina pectoris, is still less frequent. It was exhibited in only 5 out of 83 cases given in Dr. Quain's Tables, and in 8 out of the 95 given in mine.

Slow pulse, that is, under 60 in the minute, is very rare. I have not had a single example amongst the cases recorded in this chapter, and have met with only one in my experience. A man aged seventy-five years, with œdema of the feet and other symptoms of weak heart, exhibited a pulse of only 30 in the minute in the recumbent posture, but perfectly regular. The sounds of the heart were somewhat dull, but free from murmur.\*

Heberden mentions two examples of slow pulse.† A gentleman of eighty years, not ill, had a pulse which was occasionally but 26 in the minute, seldom above 30, and once only to his knowledge amounted to 42. He saw another person whose pulse at the beginning of illness was represented as only 12 to 16. But, in reference to such examples of extreme slowness of pulse, he judiciously remarks, that the radial pulsations do not represent fully those of the heart.

Doctor James Johnston gives the case of a gentleman, aged seventy-one years, whose pulse was very slow, and who after-

\* *Vide* "Rhythm of the Heart's Action," by the Author, *Dublin Quarterly Journal of Medicine*, August, 1865.

† *Medical Transactions of the London College of Physicians*, 1786, vol. ii., p. 18.

wards died suddenly. The heart was soft and brittle, but the great vessels were healthy.\*

Doctor Spens reports the case of a mechanic, aged fifty-four years, subject to headache and to attacks of syncope and convulsions, whose pulse was ordinarily from 23 to 26, and some hours before death, which took place suddenly, fell to 10 and 9 in the minute. The brain was found soft and anæmic; there was a good deal of serum in the ventricles and meshes of the pia mater. There is no notice of the state of the heart.†

A gentleman of sixty-four years had fainting fits, followed by slow pulse, not exceeding 24. On one occasion it fell to 10, 9, 8, and  $7\frac{1}{2}$ , as registered by Dr. Mitchell and Mr. Cullen. Failure of the pulse preceded the attacks of syncope, which were attended with convulsive twitchings of the face, and foaming at the mouth. Autopsy by Mr. Liston: left ventricle thin, and both auriculo-ventricular openings large; the lining of both ventricles was thick and opaque.‡

Rouchoux mentions that the First Napoleon's pulse was habitually only 40, and weak.§

Mr. B. W. Richardson reports || the case of a gentleman, aged seventy-eight years, who was also seen by Mr. Adams and Dr. Stokes, whose pulse was only 38, and afterwards fell to 24. He had had repeated attacks of syncope with dyspnœa. The feet were swollen; there was no *arcus senilis*. On examination of the body, the heart was found large and flabby. There was a large deposit of fat upon its surface, and its substance was in a state of fatty degeneration. The aorta and the coronary arteries were atheromatous; the brain was not examined.

Mr. Richardson reports another case, in which the pulse varied from 32 to 40. The patient, who was aged seventy-three years, had been subject to dyspnœa and fainting fits, not followed by paralysis. There was œdema of the feet, albuminuria,

\* *Memoirs of the Medical Society of London*, 1787, vol. i., p. 376. See also Mr. B. W. Richardson, *Dublin Journal of Medicine*, vol. xii., 1852.

† *Medical Commentaries*, 1792, *loco citat.*

‡ *Medico-Chirurgical Transactions*, vol. xxiv., p. 73. See Dr. Richardson, *loco citat.*

§ *Dictionnaire de Médecine*, "Pouls."

|| *Loco citat.*

visible pulsation of the arteries, and *arcus senilis*. There was likewise a systolic murmur both at the apex and base of the heart. Death was caused by effusion into the serous cavities. The heart was found large and soft, exhibiting both fatty growth and degeneration. The aorta and its principal branches, and the coronary arteries, were atheromatous. The mitral and the aortic valves were calcified, rough, and incompetent.

Doctor Stokes gives a list of seven cases of permanently slow pulse observed by himself, or quoted from Dublin physicians. He draws special attention to the combination of slow pulse and a tendency to syncope, with disease at the orifice of the aorta. In reference to his cases he says: "In five, organic disease of the aorta, or the valves, or both, was discovered on dissection, and in four a manifest aortic murmur existed. In two of the cases the second sound was normal, and in two there was the murmur of regurgitation in the aortic valve."\* Thus it would appear that, in the opinion of Dr. Stokes, the cause of slow pulse is to be sought primarily in the heart and aorta, and secondarily in the state of the cerebral circulation.

Doctor Law mentions† the case of a nobleman who had been subject to syncope, and whose ordinary pulse was only 25. On examination of the body, the arachnoid membrane was found opaque and thick, the pia mater infiltrated with serum, the convolutions of the left cerebral hemisphere had shrunk to half their normal size, and the cerebral substance, both grey and white, softened over one-third of the hemisphere, and nearly to the depth of the ventricles, which were full of serum. The arteries at the base of the brain were atheromatous. The pericardium was partially adherent to the heart; there were ossific plates in the mitral valve, which, however, was competent; and the aortic valve was thickened, partially ossified, and incompetent.

Doctor Quain gives 8 examples of slow pulse amongst his cases. Of these, the slowest was 24 in the recumbent and 32 in the sitting posture.

Doctor Ormerod regards slow pulse as characteristic, in an

\* *Dublin Quarterly Journal of Medical Science*, new series, vol. ii., August, 1846.

† *Dublin Journal*, vol. xvii., May, 1840.

especial manner, of fatty degeneration of the heart. In this opinion I am sure he is right; but the cause must be sought rather in the consequences of a weak left ventricle and atheromatous arteries, than in these conditions themselves; namely, in enfeebled innervation of the heart from defective nutrition of the cerebro-spinal centres and cardiac ganglia. It is worthy of remark that, in most of the reported cases of slow pulse in which a careful dissection has been made, the coronary arteries were extensively diseased, and the substance of the brain soft and anæmic; whilst on the other hand, either of these lesions singly may be commonly met with, unassociated with slow pulse.

Doctor Todd declares that hearts weak by anæmia, or fatty degeneration, may pulsate *more slowly* in the sitting or the standing, than in the recumbent posture of the body; and he expresses the opinion that this is due to the overloading of a weak heart in the erect posture. Moreover, he thinks that the equality of rate of pulsation in all postures of the body, noticed by Graves in hypertrophy with dilatation of the heart, was due to the same cause.\* But, manifestly, if this were so, pulsation should in the latter, as in the former cases, be slower in the sitting than in the recumbent posture; I have never met with an instance of the kind mentioned by Dr. Todd.

Irregularity and intermittence of the action of the heart are kindred phenomena, and due to deranged rather than to imperfect innervation. They are not, however, like slow but regular action, pathognomonic of fatty transformation of the muscular fibres of the heart, for both are frequently met with where no such transformation, and, indeed, no tissue-change whatever of the heart exists. But, whilst irregularity of the arhythmical or non-periodic kind is never unassociated with organic disease of the heart, simple intermittence very often, and indeed most frequently, is due to other causes; for example, general debility from any cause, syncope from sudden loss of blood, profound dejection, and certain chronic diseases of the brain. Irregularity in its typical form is, therefore, in general significance, a much more formidable sign than intermittence. Rhythmical irregularity, or simple derangement of *rate* in the heart and pulse is

\* *London Medical Gazette*, 1851, vol. xlvii., lecture xx.



a purely nervous phenomenon, and forbids the assumption of organic disease. Syncope, though primarily due to temporary failure of the heart, results immediately from anæmia of the brain. It usually occurs whilst some physical effort is being made which overtaxes the strength of the heart; or shortly afterwards, from the resulting fatigue. A very common cause is lifting the arms above the head, as in adjusting window curtains, hanging pictures, etc. I have repeatedly known syncope to occur where fatty degeneration of the heart existed, from the simple effort of walking in the streets. Of this, several examples will be found in the appended cases. Vertigo is an allied symptom, and due to the operation, but in a less degree, of the same cause; namely, failure of arterial circulation in the brain.

The remarkable symptoms of pseudo-apoplexy and syncopal convulsions, of which several examples are given in the list of cases, are the highest expression of cerebral anæmia. They are characterized by brief duration, frequent repetition, and recovery of the patient without paralysis. In one of Mr. Adams' cases, no less than twenty such fits occurred within the last seven years of the patient's life; and in another, four such occurred in one year. The features in these fits, which not unfrequently occur after a full meal, are usually pallid and tranquil. Sometimes, however, they are congested and livid, the difference being manifestly due to the existence in the latter contingency of spasm of the muscles of respiration generally, or of those of the constrictors of the glottis in particular. Mr. Adams is of opinion that superficial congestion may arise from obstruction at the heart, consequent upon suspension of its movements; and that to cerebral congestion arising from the same cause, may be due in many cases the apoplectic symptoms. I think, however, the two forms are primarily identical. The difference between the cases of simple syncope with pallor and loss of consciousness, and those of an apoplectiform character, with lividity of the face, turgescence of the veins of the neck, and stertorous breathing, are due to the fact, that in the latter, which are of a more aggravated character, the respiratory centre is completely anæmiated, and convulsions, with spasm of the glottis and suspended respiration, are the necessary consequences.

*Rhythmical irregularity* of breathing, under the form of alternate acceleration and suspension, with intermediate stages of ascending and descending transition from a state of the most aggravated dyspnœa to one of apnœa, is a very common symptom in connexion with fatty degeneration of the heart. This remarkable phenomenon was first described by Dr. Cheyne, in his report of the well known case already referred to (p. 601). He says: "For several days his breathing was irregular; it would entirely cease for a-quarter of a minute, then it would become perceptible, though very slow; then, by degrees, it became heaving and quick; and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration."\*

Doctor Stokes was the next writer who specially adverted to this symptom, which he very appropriately designates "respiration of ascending and descending rhythm." He refers to it as "a form of respiratory distress peculiar to this affection (fatty degeneration of the heart), consisting of a period of apparently perfect apnœa, succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the commencement of another apnœal period."

In describing the case of a gentleman, aged sixty years, who exhibited a systolic basic murmur, very loud at the top of the sternum, and who was suddenly attacked with this peculiar form of dyspnœa, which at first returned at irregular intervals, but was constant for the last two months of his life, he says: "After a period of suspended breathing, leading his attendants to believe that he was dead, a very feeble, indeed barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high, heaving, and even violent breathing was established, which would then subside until the next period of suspension. This was frequently a-quarter of a minute in duration." He adds: "I have little doubt that this was a case of weakened and probably fatty heart, with disease of the aorta."†

\* *Dublin Hospital Reports*, vol. ii., p. 216, 1818.

† *Dublin Quarterly Journal*, vol. ii., new series, August, 1846.

In this opinion, although no opportunity was afforded for confirming it by dissection, he was most probably correct. I believe however, that this remarkable symptom, of which several typical examples will be found in the annexed cases, although in the great majority of examples associated with the twofold lesion mentioned by Dr. Stokes, depends, not upon the fatty state of the heart, but upon atheromatous change, dilatation, and loss of elasticity in the aorta. In numerous examples of veritable fatty transformation of the heart, verified by *post mortem* examination, included in my records, this symptom was *not* exhibited; but in these the above mentioned changes in the aorta had not taken place; whereas, of the many cases in which rhythmical irregularity of breathing was present, *not one* failed to present this condition of the aorta, combined with fatty degeneration of the heart, where the actual state of the organs was determined by dissection.

In illustration of the rhythm of this form of dyspnoea, I may select the three following examples.

A man, aged sixty-two years, was suddenly seized with dyspnoea whilst proceeding to his work. Since then he had been subject to these attacks, which were accompanied with the most aggravated suffering. He felt as if his breathing were about to cease. Whilst he lay in bed, his breathing would gradually become quick and gasping, till it attained a rate of 63 in the minute, and then as gradually subside till it ceased entirely, continuing suspended for twenty-two seconds. During the period of apnoea there *was no change* in the heart's action or radial pulse, in regard to rate or otherwise (see Case 51, James K.)

A man aged sixty years, was subject to dizziness and paroxysmal dyspnoea, during which there were alternately periods of apnoea and suspirious respiration, the former occupying twenty-four, and the latter twenty-six seconds, the transition being rather abrupt and occupying only about one second. The action of the heart and the pulse *underwent no change* during this revolution of breathing (see Case 52, James D.).

A man of large stature, a plumber by trade, of temperate habits, suffering from general dropsy and the symptoms and

signs of hypertrophy and fatty degeneration of the heart, dilatation and roughening of the aorta, and disease of both the aortic and mitral valve, exhibited in the most typical form respiration of this character. The following description of this phenomenon is taken from my notes, made when the case was under observation, February, 1872.

“There was a period of complete suspension of breathing, which, in the recumbent posture, lasted fifteen seconds; during its continuance the poor patient, worn out by want of sleep, seemed to enjoy complete respite from suffering, and passed into a calm slumber. Then a shallow inspiratory movement of the chest was observed; another, somewhat deeper, followed at an interval of about two seconds; respiration became now progressively deeper and more rapid, till after about a dozen acts it had attained the character of a paroxysm of great severity, accompanied by a deep and distressing moan. After about a dozen such quick respiratory efforts, extending over a period of as many seconds, moaning ceased, and the breathing became gradually less and less deep and rapid; till, at the end of about fifteen seconds, it was entirely suspended, and another and similar cycle was commenced. During the paroxysm of dyspnoea the heart's action was remarkably irregular.” (See Case of C. D., aortic disease.)

Respiration of this character existed in 29 cases included in my Tables; viz., in 16 of Table V.; in 1 of Table VI.; and in 12 of Table VII.\*

Amongst Dr. Quain's cases I have not found a single example of the kind recorded, although dyspnoea is represented as present in thirty-four instances.

Is fatty transformation of the muscular fibres of the heart necessary to the existence of this symptom? Dr. Stokes holds that it is, whilst I regard it as only accessory; the essential condition being, in my judgment, dilatation and loss of elasticity in the aorta, with which, however, fatty change of the heart is almost invariably associated.

Doctor Seaton Reid, of Belfast, published, in 1850, a case in which this symptom existed, and the muscular structure of the heart was found healthy. There was, however, dilated hyper-

\* *Vide* p. 648, *et seq.*



trophy of the left ventricle, dilatation with atheromatous change of the aorta, and incompetence of the aortic and mitral valve.\*

Doctor Head communicated to the Medical Society of the College of Physicians, in the session 1867-8, another instance of a heart free from fatty degeneration, where this symptom in a typical form had been exhibited, the lesions present being aortic patency and hypertrophy of the left ventricle.

Doctor James Little, in 1868,† published three cases, in each of which "ascending and descending" respiration existed. In one of these, that of a man of sixty-nine years, fatty degeneration of the heart was found, but no valvular disease. In another, who was aged seventy-two, the left ventricle was hypertrophied, the aortic valves slightly rigid but not incompetent, and the aorta atheromatous and calcareous; and in the third, who was also seventy-two years old, the left ventricle was much dilated and hypertrophied, the aortic valves thick and rigid, but competent, and the entire arch of the aorta was dilated and atheromatous. The muscular structure of the heart was absolutely free from fatty degeneration, as reported by Professor Macalister, a most competent authority.

Thus it would appear that fatty degeneration of the substance of the heart, though usually present, does not constitute a necessary condition of the existence of rhythmical irregularity of breathing; whilst, on the other hand, in all the examples of it which have come under my notice, or of which I have seen the report, the primary portion of the aorta has been atheromatous and dilated.

Doctor Seaton Reid was the first to observe and record a change in the rate of the heart and pulse, inversely as those of respiration. He stated that during the period of accelerated breathing, the pulse became invariably slow, increasing in rate during that of apnoea.

Doctor Little also observed this phenomenon in two of his cases. He says: "In a note made on the 14th August, it was recorded that during ten seconds of the breathing period, there were only six systoles of the heart, and these were generally

\* *Dublin Hospital Gazette*.

† *Dublin Quarterly Journal of Medical Science*, No. xci.

irregular in rhythm and force, while, during ten seconds of the apnoeal period, there were fifteen; the sounds were indistinct." Again, in reference to his third case, he says: "From a note made two days after his admission, I find that the apnoeal period lasted ten seconds, and during that time nineteen radial pulses were counted. The respiratory acts occupied twenty-five seconds, and during ten seconds, counted off at the height of the paroxysm, thirteen systoles were felt at the wrist."

I have not, in any of my cases, met with this change in the rate of the heart and pulse; on the contrary, I have specially and repeatedly noted the unaltered rate and character of both, during the changes of rhythm and force constituting this remarkable phenomenon.

Doctor Little propounds\* the following theory in explanation of this symptom: "In health, the right and left ventricles, though differing so much in the thickness of their walls, are equally competent for their duties; the right is able to fill the pulmonary capillaries as thoroughly as the left, with the aid of the other forces which contribute to the circulation, fills the systemic. But if an abnormal burden is imposed on the left, if rigid valves narrow its outlet, or permit the blood it discharges at each systole to fall back into its cavity, or if the arterial coats, their elasticity destroyed by disease, no longer help the heart; if the aorta, instead of taking charge of each wave of blood as it leaves the ventricle and propelling it onward by the steady recoil of its walls, is permanently dilated, and allows each portion of blood to remain in its ascending trunk, and so to impede the entrance of that which follows—under any of these conditions, the left heart, however hypertrophied, may be quite unable to rid itself of the blood as rapidly as it is supplied to it by the right ventricle. Blood would, therefore, accumulate in the left auricle, in the pulmonary veins, and in the capillaries of the lungs. That blood, having already absorbed as much oxygen as it required, would fail to produce that impression on the ultimate filaments of the pneumogastric which black blood does, and which impression is converted by the nervous centres into the motor impulse which produces breathing. Breathing would,

\* *Leco citat.*

therefore, cease; and, inasmuch as the respiratory act seems to assist in carrying the blood to the left side of the heart, it would no longer be so over-stimulated by fresh supplies, and its contractions would become less frequent and more regular. After a few systoles, however, it would succeed in discharging the red blood collected in its cavities to such an extent that they could receive some of that which lay in the pulmonary veins and lungs. Space being thus gained, the black blood which the pulmonary artery contained would reach the capillaries of the lungs in amount proportionate to that of the arterial which had gone forward, and sufficient air would be drawn into the chest to aërate so much blood. That very act would carry forward a still larger charge of arterial blood to the left side, and make room for the reception, by the lungs, of a still further increase of venous blood; and, as a consequence, a still deeper inspiration would follow, and the deepest would occur when the largest quantity of venous, and the smallest quantity of arterial, lay in the lungs. The red blood, reaching the left heart, would excite it to those frequent and irregular contractions which accompany the respiratory distress, but frequent and irregular, they would be also ineffectual; red blood would begin again to accumulate in the left heart, the pulmonary veins, and the lungs; till at last their capillaries would contain little else, and the exciting cause of inspiration, the venous blood, being no longer present, the act itself would again cease."

From the preceding quotation, it would seem that Dr. Little's very ingenious theory is based upon the assumption, that to derangement of dynamic adjustment between the right and left ventricle of the heart, is primarily and essentially due the phenomenon of respiratory disturbance. There are, however, certain difficulties in the way of its acceptance, which I shall here content myself with stating. The principal of these Dr. Little has anticipated; namely, that whilst derangement of equilibrium between the two sides of the heart, by enfeeblement, actual or virtual, of the left ventricle, is very common, respiratory distress of this peculiar character, is comparatively rare. For example; advanced fatty degeneration of the left ventricle, the right being very slightly, if at all, affected, is not uncommon.

Neither is attenuation of the left ventricle without a corresponding change in the right, in old, ill nourished, and hard worked persons. Yet in neither of these cases, where other and very palpable changes have not taken place, is the phenomenon in question witnessed. Again, lesions of the aortic valves, involving obstruction and inadequacy, and obstruction at the mitral orifice, which is no less effectual in the production of arterial blood-stasis in the lungs, though of frequent occurrence, are rarely associated with this symptom, and never so associated in the absence of other changes.

Finally, the assumption involved in this theory, that the *besoin de respirer* has its seat in the air vesicles of the lungs, and for its cause, the reflex irritation arising from the presence in them of carbonic acid, is unwarranted. The experiments of Flint\* have clearly proved that the feeling of the "want of air" has its seat, not in the lungs exclusively, but in the tissues of the body generally; and that the excitant of that feeling is not carbonic acid by *its presence*, but oxygen by *its absence*.

I have already stated that the only lesion of structure with which rhythmical irregularity of breathing has been always found associated, is atheromatous or calcareous change, with dilatation, of the arch of the aorta, involving loss of elasticity in its walls. I think these changes supply the conditions of a rational theory of the phenomenon. During the period of greatest tranquillity of the heart's action, viz., in sleep or repose, the systemic capillary circulation fails, from want of the contributory aid rendered in health by the elastic reaction of the aorta. Hence arise a suspension of tissue-respiration, *besoin de respirer*, and accelerated or suspirious breathing, as shown by the experiments of Flint already referred to. Accelerated respiration must strengthen capillary circulation; first, through the lungs, and then through the tissues of the body generally, by quickening the action of the heart, and increasing its force. In proportion as the systemic capillary circulation becomes established, the *besoin de respirer* is less urgent, and respiration gradually subsides, till a period of apnoea arrives. The descent of respiration below the normal standard would seem to arise from its previous excessive activity, and the ex-

\* *Physiology of Man*, article "Respiration," 1866.



haustion of the patient. Now, again, comes a period of feeble action of the heart, and failure of capillary circulation, with its consequence of paroxysmal breathing. That imperfect circulation of arterial blood in the respiratory centre contributes in a special manner, and in a great degree, to the production of the respiratory derangement, I have no doubt; but the effect of this is not easily distinguished from that of a want of oxygen in the tissues of the body generally. The *fact* that dilatation and rigidity of the arch of the aorta is, *par excellence*, the lesion with which rhythmical irregularity of breathing is associated, and of which it may, therefore, be regarded as pathognomonic, will be in no degree affected by the estimate formed of the foregoing theory. This theory is propounded only with a view of presenting in the form of a connected and rational series the subphenomena constituting this remarkable symptom.

Doctor Little observed that the respiratory distress became gradually less urgent, and finally ceased, at the near approach of death.

I have not observed this in any of my cases. Theoretically, I would have expected it, irrespective of the positive testimony of so competent an observer as Dr. Little. Yet its absence in the cases which have come under my notice deserves to be mentioned, as showing that where the respiratory phenomenon under consideration constitutes a feature of the final illness, it may continue up to the moment of death. Indeed, in a few instances it seemed in no inconsiderable degree to aggravate the last sufferings of the poor patient.

Neither have any of my cases exhibited the marked change in the rate of cardiac pulsation, at the different periods of the paroxysm, which Dr. Little has noticed. I have, on the contrary, as already stated, observed either an absolute uniformity of the pulse throughout the attack, or, as in one or two cases, a slight acceleration during the period of dyspnoea, and a gradual decline in that of descent, till a minimum rate was reached on the accession of apnoea.

In 1864, and again quite recently,\* Professor Laycock expressed the opinion that this phenomenon depends upon "a

\* *Dublin Journal of Medical Science*, July, 1873.

sentient palsy of the respiratory centre," or "a paresis of reflex sensibility of the mucous membrane of the lung;" that "the slower breathing is due to the diminishing sensibility, and the accelerated breathing following the interval of apnœa is due to the stimulus of unaërated blood being hurried proportionately to the need of oxygen." It is, he thinks, "a neurosis of the vagus, not necessarily dependent on structural or other diseases of the heart," and most frequently occurs during sleep, or at the moment of going to sleep; showing that "the gradually diminished motor activity is coincident with gradually diminished sensory activity, until the carbonized blood rouses up the sensory centre."

That lowered sensibility of the vagus centre is a prominent factor in this phenomenon, I am quite in accord with Professor Laycock in holding; but I cannot agree with him in the opinion that it is not necessarily connected with structural disease of the heart, at least, when the latter is associated with disease of the aorta. It is conceivable that diminished sensibility of the vagus centre should be a not unfrequent consequence of one or more of the various forms of organic disease or injury of the brain. It is certainly an associated condition in coma, to whatever cause due. Yet, rhythmical disturbance of breathing is not a symptom of cerebral lesion, or of the comatose state. Starting from sleep, especially when lying on the left side, and in a state of great alarm and dread of some indefinite evil, are usually associated with rhythmical derangement of respiration. The tongue is generally loaded, the stomach distended with flatus, and the bowels constipated, no doubt owing to congestion of the portal system.

Dropsy is not a proper symptom of fatty degeneration of the heart, although it not unfrequently is associated with that affection; but, primarily so, only when the kidneys and liver are likewise engaged. In the advanced stages it is often present, not from the special tissue-change in the heart, but from weakness and dilatation consecutive to it. It existed, in some form, in 25 out of 95 cases in my tables; or, in somewhat more than one-fourth of the whole; that is, in 5 cases, in Table V.; in 8, in Table VI.; and in 12, in Table VII.\*

Doctor Ormerod noted it in 10 of his 25 cases.

\* *Vide* p. 648, *et seq.*

I may, however, remark, as a clinical fact of which I do not venture to offer an explanation, that œdema of the lower limbs, and extending to the external genitals, is eminently suggestive of a fatty state of the heart and kidneys conjoined. I have not met with a single instance of it, in which, on dissection, this twofold lesion was not found.

Granular degeneration of the kidneys existed in 7, and fatty degeneration of these organs in 11 instances out of the total of 95 cases in my tables; and in those of Dr. Quain, comprising 83 cases, the numbers were 9 and 2 respectively.

The liver is usually engorged in the advanced stages of fatty degeneration of the heart, owing to weakness and dilatation of the right chambers. There is, then, fulness and tenderness on pressure at the epigastrium, and the liver may be felt descending to a greater or less distance towards the umbilicus. But occasionally the liver is primarily engaged, either by cirrhosis or fatty change. The former was witnessed in 4 instances out of the 95 cases included in my tables; and the latter in 12. Where jaundice occurs, the condition of the liver is not that of simple engorgement; and where, with this symptom present, cirrhosis is excluded, fatty liver may be diagnosed.

Arcus senilis is regarded by Mr. E. Canton as pathognomonic of fatty disease of the heart.\* He declares that he has "in no instance found this senile arc, when well developed, unaccompanied by fatty degeneration of the heart; and further, that "the extent of change in the cornea may be regarded as a measure of the degree to which the heart-fibre has, in the same manner, become changed." He adds that Dr. C. J. B. Williams had found the "arcus" in 23 cases out of 25 of fatty heart. Of this number, however, the diagnosis was verified by *post mortem* examination in only 2 instances.

Doctor Fuller regards this sign "as of little value in the diagnosis of fatty degeneration of the heart."† And Doctor Haskins gives a list of 12 cases of "arcus," in only 2 of which were there any symptoms referable to the heart; and in 1 only could any organic alteration be suspected; 6 out of the

\* *Lancet*, May 11th, 1850, p. 561; and January 18th, 1851, p. 67.

† *Diseases of the Chest*, 1862.

12 patients were perfectly healthy.\* This evidence, though strong, is not conclusive, in the absence of proof by dissection, that fatty change of the heart did not exist. Arcus existed in 8 only of the 95 cases included in my tables; viz., in 6 of those in Table V., and in 1 in Table VI., and Table VII., respectively.†

Doctor Milner Fothergill regards the dim and hazy form of "arcus," with ill defined edges, fading off into a cloudy cornea, as pathognomonic of degenerative changes, and eminently suggestive of a state of fatty change of the heart.‡ Whilst admitting that a cloudy ring in the outer margin of the cornea, positively indicates fatty decay and disintegration of that structure, and suggests similar changes in other tissues and organs, I am not prepared to concede that those tissues must necessarily be muscular, and the organ engaged, the heart.

I cannot, therefore, accept either of Mr. Canton's propositions; namely, that the heart-substance must necessarily be in a state of fatty degeneration where well formed "arcus" exists; and that the latter may be taken as a measure of the former condition.

Where strong and direct evidence of a morbid condition of the substance of the heart existed, I should consider the presence of arcus senilis as warranting the presumption that the tissue-change in progress was fatty degeneration. This condition, when present, is not necessarily universal as regards similar structures, much less as regards those which are histologically dissimilar. The liver alone may be the seat of fatty change; the muscles of one or more of the limbs, or even a particular group of congenious muscles, may alone be found to have undergone fatty degeneration.

It is no less certain that the corneæ may exhibit the fatty arc, without the heart being in a similar state of histological change; and, again, that the heart may be the subject of fatty degeneration when no arcus senilis exists. The "arcus," in short, may be regarded as a presumptive, but not as a positive symptom of fatty degeneration of the heart.

The *physical signs* are individually inconclusive, but collec-

\* *American Journal of the Medical Sciences*, January, 1853.

† *Vide*, p. 648, *et seq.*

‡ *The Heart and its Diseases*, 1872, p. 170.



tively they are of positive significance. Of these, the most important have reference to the sounds of the heart. The first sound is always short and faint. In the early stages it is likewise dull and ill pronounced, the impulse element, though feeble, masking the valvular in different degrees. When fatty degeneration is secondary to hypertrophy, the valve-click is completely muffled at the apex; but when, on the other hand, the tissue-change is primary, the walls of the heart being of normal or reduced thickness, the valve-element of the first sound is faintly audible.

In the advanced stages of primary fatty degeneration, the first sound so closely resembles the second, that, in the absence of cardiac impulse, it becomes necessary to place the finger on the carotid in order to determine its rhythm. This is manifestly the result of a twofold cause; namely, the weakness of the heart, by which the impulse-element is suppressed, and the attenuation of its walls, through which the valve-sound is conducted more distinctly to the ear. Where attenuation has not existed at any stage of the affection, as in hypertrophy undergoing the fatty change, the first sound is dull at the apex, and sharp and valvular at the base, throughout.

Out of the total of 95 cases in my tables, the first sound was dull in 16; *i.e.*, 5 out of 55 in Table V.; 7 out of 18 in Table VI.; and 4 out of 22 in Table VII. The second sound was sharp and clear in 7 instances; *viz.*, 2 in Table V., 1 in Table VI., and 4 in Table VII. I have, in a few instances, found mitral regurgitant murmur of a soft and blowing quality, and adynamic origin, in connexion with fatty degeneration and softening of the left ventricle; the valves, on dissection, were discovered to be perfectly sound and competent. This I am obliged to attribute to yielding of the parietal attachment of the papillary muscles at the acme of ventricular systole.

From dilatation with thinning of the walls of the left ventricle, without structural change, as exemplified in middle aged, hard worked, ill fed and fretful subjects, chiefly females, fatty degeneration with parietal thinning, may, in most instances, be readily distinguished by the physical signs alone, and even where the distinction is not practicable by reference to the history and general symptoms.

In dilatation with thinning, the sounds are sharp, ringing, of normal length, remarkably loud, and extensively transmitted over the chest both before and behind. A distinct apex-pulsation is detectable by the hand to the left of the ordinary position, characterized by a swell of gradual elevation and subsidence, but without tension. The pulse is weak and regular, varying in rate under slight excitement or emotion, and not unfrequently intermitting at distant intervals. The patient is anæmic, and, with the exception of dyspnoea consecutive to intercurrent pulmonary congestion, to which the patient is liable on slight exposure, and this of the ordinary character, there are present none of the symptoms which specially belong to fatty disease of the heart.

In the latter disease, the first sound at the apex is, on the contrary, either masked or sharp according to the thickness of the walls of the left ventricle ; but it is always brief and faint, and not audible beyond the precordium, except feebly in the course of the ascending aorta, and precordial impulse of any kind is rarely perceptible.\* It is unnecessary to specify the other symptoms already discussed, as characteristic of fatty degeneration of the heart ; viz., irregularity or slowness of pulse, pseudo-apoplectic seizures, syncope, rhythmical irregularity of breathing, and arcus senilis.

Doctor Fuller deems it impossible during life to distinguish between simple softening, fatty softening, and attenuated dilatation.† I cannot admit simple softening, as distinct from typhoid or fatty softening. Typhoid softening is readily distinguished by the rapid development of its proper signs, and by the coexistence of fever of the typhoid type. Attenuated dilatation may, I think, be readily diagnosed by the rules just stated.

The diagnosis of fatty degeneration consecutive to hypertrophy, and especially the determination of the period when this change is inaugurated, are of much consequence ; because of the extreme gravity of this form of the affection, and the advantage

\* Doctor Stokes remarks that occasionally, in cases of extreme fatty degeneration of the heart, a diffused impulse may be felt without apex-beat, resembling the heaving of a thin-walled aneurism. *The Diseases of the Heart and Aorta*, p. 328.

† *Opus citat.*

of an early application of hygienic and therapeutic measures, with a view to the arrest of tissue-decay.

The characteristic symptoms of this change are implied in the expression, *failure of the heart*. Thus, one of the earliest symptoms, indeed in one class of cases the earliest, is tumultuous action or palpitation, with feeble and irregular pulse, when the patient makes any sudden effort; it is most likely to occur if the patient rapidly ascend a steep incline, such as a flight of stairs, or hurry over a journey on foot, being urged forward by a desire to be up to time. Such is usually the first indication of "break up" in those who at the middle period of life, have enjoyed good health, with the exception of occasional twinges of gout, and have been of active habits, and lived freely; or, if of the working classes, who have indulged in the use of malt drink. An eminent medical practitioner in a country district, of middle age and full habit, informed me that the first intimation which he received of disease of his heart, consisted in palpitation, precordial oppression, and overpowering dyspnœa, on rapidly ascending a flight of stairs in his own house, in order to procure some medicine before answering a hurried call. He was ever afterwards subject to paroxysms of dyspnœa, which gradually became more frequent, and would ultimately attack him even when at rest. I attended this gentleman in his last illness, which was characterized by all the symptoms and signs of enlarged and fatty heart, with atheromatous and dilated aorta. (See Case 47, J. N. W.)

The case (51), previously referred to (p. 627), affords another example of the same kind. A working mechanic, over sixty years of age, who had lived well, but temperately, noticed for some time slight shortness of breath on exertion. He received a severe shock from the fall of a beam of wood upon him, and was thenceforward subject to more severe accessions of dyspnœa. A week previous to my seeing him, he was proceeding hurriedly to his work after his morning meal, when he was suddenly seized with dyspnœa and oppression, so urgent that he was forced to return home, and was never afterwards able to resume his labour. There was present all the evidence of enlarged and fatty heart, dilated aorta, and disease of the mitral and aortic valves, which was subsequently confirmed by dissection.

The history of all such cases is that of atheromatous aorta, with or without valvular disease and hypertrophy, and, subsequently, fatty degeneration and dilatation of the left ventricle.

The two last mentioned changes are those which determine the symptoms of failure of the heart. When fatty degeneration supervenes upon hypertrophy from valvular disease at the aortic or mitral orifice, although the "break up" of the general health is more or less abrupt, and usually attributed to a definite cause involving muscular effort, still, as in the preceding example, the surprise is not complete; because, from the previous existence of symptoms referable to the valvular lesion, the patient must have been sensible that he was the subject of some form of cardiac affection. Here likewise, however, failure of the heart from fatty degeneration and dilatation is the immediate cause of the critical change. When symptoms of this critical character have been exhibited, it is too late to attempt anything with a view to arresting the progress of the disease. But if the patient had been previously under skilled observation, it would be the duty, as no doubt it would be within the competence of a well informed practitioner, to determine from the occasional presence of vertigo or amblyopia, irregularity of pulse, and slight feeling of suffocation with hurried breathing, the incipience of fatty degeneration of an hypertrophied heart. Then it is, that by regulating the diet and habits of the patient, and putting him promptly under a suitable course of medicine, the process of retrogressive change may be retarded, or even arrested.

In other and not fewer instances, the pathological series commences with granular degeneration of the kidneys, accompanied not unfrequently by atheromatous change in the coats of the aorta. Then follow in succession hypertrophy, fatty degeneration and dilatation of the left, and next, of the right ventricle, and fatty conversion of the kidneys themselves. In this latter case, cedema of the feet is usually the first, as effusion into the serous cavities constitutes the last, member of the series. These are usually the most urgent and distressing cases; they are not unfrequently complicated with erysipelatous inflammation and sloughing of the lower limbs. The presence of albuminuria, with a low specific gravity of the urine, would here constitute the first symptom of



disease ; but, as in the former case, the earliest indications of fatty change in the heart would have reference to its failure. Where fatty degeneration is secondary to inflammation of the heart, and not preceded by hypertrophy, the substance of the heart has been the seat of the inflammatory process. In other words, of the phlegmasiæ of the heart, myocarditis alone is capable of leading directly to fatty transformation of its substance. This latter is the most rapidly fatal process of fatty change to which the heart is liable, because of the absence of the protective influence of hypertrophy.

The heart was hypertrophied in 39 out of a total of 68 cases of fatty degeneration reported by Quain. It was normal in 8, and atrophied in 4 instances. In my list of 95 cases, the heart was hypertrophied in 31 instances, and dilated in 37.

Doctor Quain inquires whether hypertrophy may not proceed "from a natural effort to compensate for loss of power." I think not ; because the antecedent state of fatty degeneration implies impairment of nutrition.

A certain incompatibility has been assumed to exist between fatty degeneration of the heart and valvular disease.

Doctor Henry Kennedy declares that out of a list of 245 cases of fatty heart, collected by him from different sources, there were only 33 in which disease of the valves existed.\* He refers to a list of fifty deaths from the administration of chloroform, published by Snow, in nearly all of which fatty disease of the heart *without* valvular lesion was found ;† and adds, that when valvular lesion exists in such connexion, it is usually located at the orifice of the aorta, the valves being not atheromatous but fatty and competent.

Doctor Walshe mentions "absence of murmur" as negative evidence of fatty heart.‡

Doctor Stokes, on the contrary, is of opinion that "valvular disease is a not infrequent combination with fatty heart." In most of the cases which he has seen, the valvular affection was at the aortic orifice ; and he calls attention to the existence of

\* *Proceedings of Surgical Society of Ireland*, March, 1864.

† *Proceedings of the Pathological Society of Dublin*, vol. i., January 8th, 1859.

‡ *The Diseases of the Heart*, third edition, 1862, p. 351.

aortic systolic murmur, either blowing or musical, accompanying or replacing the first sound, the second sound being either normal or suppressed, but *without* the signs of regurgitation, as confirmatory evidence of fatty heart. He admits only two forms of valvular lesion in connexion with a fatty state of the heart; viz., those causing aortic, and mitral obstruction.\*

Jaccoud tries to establish a special connexion between aortic valve-inadequacy and fatty degeneration of the heart; urging that, inasmuch as the coronary arteries receive their charge of arterial blood during diastole, regurgitation at the aortic orifice must diminish both the volume and the pressure of the coronary circulation, and must consequently lower the nutrition of the heart. He maintains, however, that the degenerative change is of a mixed or "fibro-fatty" character, relying upon the authority of Billroth and Traube, who assert that muscle tends to degenerate into connective tissue whenever its nutrition is impaired. He thinks it probable that, in such cases, myocarditis progressed concurrently with the endocarditis which disorganized the valves, and favoured the occurrence of tissue-degeneration.† With regard to this statement, I may observe that I have not investigated the matter specially in relation to aortic reflux; but in ordinary fatty degeneration, even that depending manifestly upon defective supply of arterial blood, I am in a position positively to deny the existence of any increase of the plastic or fibroid element. In reference to the assumed origin of the valvular lesion from endocarditis in such cases, and the argument founded thereon, I will only remark that, in my opinion, this lesion is rarely of inflammatory origin, and is most frequently of a degenerative type, like the fatty change in the heart itself, and is coeval with it. A "disorder of compensation," such as hypertrophy of the left ventricle consequent upon aortic insufficiency, may be converted into "derangement of compensation." When the latter is due to a temporary cause, such as fatigue, or bronchitis, it is not of grave augury; but when it results from the natural progress of the disease, as exemplified in mitral incompetence from fatty softening and dilatation of the

\* *The Diseases of the Heart and Aorta*, 1854. p. 330-1.

† *Medical Press and Circular*, July, 1868, translated by G. Cockle, M.D.

left ventricle, consecutive to hypertrophy from incompetence of the aortic valve, then the prognosis is very grave, and a state of permanent asystolie is established. On this subject, Tables V., VI., and VII. (pp. 648, 656, and 660), show the following results :

Condition of Valves.	Table V. 55 Cases.		Table VI. 18 Cases.		Table VII. 22 Cases.		Total. 95 Cases.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
Valves normal ..	16	28·80	12	66·60	9	40·90	37	38·95
Aortic valve diseases ..	9	16·36	1	5·50	8	36·36	18	18·90
Mitral valve diseases ..	6	0·90	..	..	9	40·90	15	15·80
Not stated ..	21	38·18	5	27·70	..	..	26	27·36
Aortic-systolic murmur	4	7·27	..	..	8	36·36	12	12·60
Aortic-diastolic murmur	1	1·81	1	5·50	2	9·09	4	4·20
Double aortic murmur	3	5·45	..	..	2	9·09	5	5·26
Mitral-systolic murmur	1	1·81	3	16·60	7	31·81	11	11·60
Left presystolic murmur	1	1·81	..	..	1(?)	4·54	2	2·10

Thus it would appear that, if the instances, 26 in number, in which the state of the valves is not mentioned, be deducted from the total of 95 cases, the residue is nearly equally divided into those in which the valves were unaffected, and those in which one or more sets of them were disorganized ; the proportion being as 37:33.

In 34 instances, murmur of some kind was present ; and of these it is noteworthy that it was located at the aortic orifice in no less than 21 instances, being systolic in rhythm in 12 of the number ; and at the mitral orifice in 13 instances, being likewise systolic in 11 examples.

In Table VI., for the items in which I am personally accountable, aortic murmur is stated to have existed in 12 instances, whereas disease of the aortic valve was actually present in only 8. In the remaining 4 cases, the murmur was produced in the aorta itself by atheromatous or calcareous roughening of its inner surface.

The practicability of diagnosing a fatty condition of the heart, especially in its early stages, has been questioned by some eminent authors. Dr. Latham denies it, and Dr. Stokes expresses a

qualified opinion in the following terms : "The fatty degeneration considered alone, does not, so far as we know, afford any special or separate sign ; and it would be difficult or impossible to draw a line of distinction between the signs of simply weakened heart, and of this condition combined with fatty degeneration." "In many minor cases of this condition, the heart's sounds may present no abnormal character."\*

It will be observed that the preceding opinion has reference to the signs exclusively. I think, however, that the absence of the impulse, or its extremely feeble character; the brief duration of the first sound, whether masked or sharp, in primary cases, and its almost complete or absolute extinction in those preceded by hypertrophy; the restriction of the sounds within a very limited area; and the occasional irregularity of the heart's action, will suffice, in the majority of cases, to establish the diagnosis of fatty heart from the physical signs alone.

The existence of a single murmur of systolic rhythm at the root, or in the ascending portion of the arch of the aorta, may be taken as strong collateral evidence of a fatty state of the heart; because of the very frequent association of this condition with atheroma of the aorta, involving the valves or not. The aorta was atheromatous in 40 out of 95 cases epitomized in my tables; nor should this be matter for surprise, atheromatous transformation of the aorta being itself a veritable fatty degeneration, as shown by Gulliver and Virchow. The absence of diastolic basic murmur affords, I think, in such cases, no proof of the competency of the aortic valves; the reaction of the aorta, owing to the loss of its elasticity from disease of its coats, being inadequate to the production of a reflux current of sufficient force to develop a murmur. If, at the same time, independent evidence of hypertrophy, with dilatation and elongation of the left ventricle, be present, as afforded by downward and outward displacement of the apex, collapsing, and visible pulse, and throbbing of the carotids, the existence of aortic regurgitation may be confidently affirmed, notwithstanding the absence of the characteristic diastolic murmur.

If the typical symptoms of occasional vertigo, momentary

\* *Opus citat.*, p. 327-8.



failure of sight, dizziness, syncope, or pseudo-apoplexy, and paroxysmal and rhythmical dyspnœa\* coincide in any given case with the physical signs just mentioned, the positive diagnosis of fatty heart would be fully warranted.

The incipience of primary fatty degeneration may be suspected, if the pulse, previously regular, become weak and irregular; if the surface be pale, the patient subject to dizziness or syncope, and the cardiac impulse feeble; although the sounds of the heart may not appreciably differ from their normal character. A positive diagnosis, however, would be impracticable under the circumstances.

The mode of death is most frequently slow, and by a gradual process of sinking or exhaustion. Less frequently it takes place by a gradual process of a different kind; namely, by asphyxia, or by coma. It may likewise occur, rapidly, from angina pectoris, from a leakage of blood into the pericardium, or from rupture of the septum ventriculorum; or, instantaneously, by simple syncope, or from rupture to a large extent of the outer wall of the heart, or of the aorta. Amongst Ormerod's and Quain's cases of fatty degeneration, 93 in number, death occurred by syncope in 10, by coma in 1, and by rupture of the heart in 22 instances. The following is a summary, in tabular form, of the mode of death in 83 out of the 95 cases comprised in Tables V., VI., and VII.:

MODE OF DEATH.

Causes.	Table V. 55 Cases.		Table VI. 18 Cases.		* Table VII. 22 Cases.	Total. 95 Cases.	
	Cases.	Per cent.	Cases.	Per cent.		Cases.	Per cent.
Asthenia .. ..	23	41·81	5	27·70	17	45	47·37
Angina Pectoris ..	8	5·45	1	5·50	..	4	4·21
Asphyxia .. ..	1	1·81	4	22·20	2	7	7·37
Coma .. ..	3	5·45	1	5·50	3	7	7·37
Rupture of heart ..	15	27·27	2	11·10	..	17	17·89
Rupture of aorta ..	6	10·90	1	5·50	..	7	7·37

With regard to the seat of rupture, in these 17 instances; it occurred on the anterior wall of the left ventricle in 8 cases; on the posterior wall of the left ventricle in 6; on the anterior sur-

\* The significance of this symptom depends upon the very common association of fatty heart with dilated and atheromatous aorta, of which it is pathognomonic.

face of the right ventricle in 1 ; through the septum ventriculorum, not communicating with the exterior, in 1 ; and through the septum ventriculorum, ultimately penetrating the anterior wall of the right ventricle, in 1 instance. In the last mentioned case the patient lived six days after the occurrence of the primary rupture.

It will thus be seen that the outer wall of the left ventricle was the seat of rupture in no less than 14 out of 17 examples of this accident, and that it occurred through the anterior, more frequently than through the posterior wall, in the proportion of 8 : 6.

Of Quain's 22 cases, rupture of the external wall of the left ventricle took place in 17 ; viz., on the anterior surface of the ventricle in 12, and on the posterior surface in 5 instances. The septum ventriculorum was rent in 1 instance, the right ventricle in 2, and the right auricle in 2 cases.

It is noteworthy, that when the septum ventriculorum was the seat of rupture, the patients invariably survived the accident several days. Such was likewise the case in the examples, five in number, of partial rupture of the wall of the heart. One such accident occurred through the deep layers of the left ventricular wall ; one through those of the right ventricle ; one in a similar situation in the right auricle ; and one through the superficial fibres of this chamber, not communicating with its interior.

The symptoms indicative of rupture of the heart, with the exception of that of the septum of the ventricles, are sudden and severe lancinating pain, accompanied with a feeling of sinking and fluttering in the region of the heart, arrest of respiration followed by a few gasping inspiratory efforts at long intervals, failure of the pulse, extreme pallor quickly succeeded by capillary venous engorgement of the cutaneous surface, loss of consciousness, and death within a period of a few minutes. When the septum ventriculorum alone has been rent, the patient usually survives several days ; and in one instance, already mentioned, a period of six days actually intervened between the date of this accident and that of death. Dr. Quain mentions a case in which the patient survived eight days.\*

\* Lumleian Lectures, *Lancet*, March, 1872.

Three of the most eminent Scotchmen of the last generation, Drs. Chalmers, Abercrombie, and Sir J. Y. Simpson, died of fatty disease of the heart; the two former suddenly. Dr. Chalmers died of simple syncope, and Doctor Abercrombie from rupture of the left ventricle. In Dr. Chalmers' case there were no premonitory symptoms, except an attack of temporary hemiplegia, from which he entirely recovered in the course of a few weeks, thirteen years before death. Dr. Abercrombie had, four years before death, an attack of faintness, with oppression and headache of brief duration, but no other premonitory symptom. The heart was in a state of fatty degeneration. Dr. H. Bennett reported on Dr. Chalmers' heart, the left ventricle of which was almost entirely converted into fat, exhibiting under the microscope lines of granules, and no transverse striation.\* Sir J. Y. Simpson died of fatty degeneration of an hypertrophied heart, but by gradual exhaustion, or asthenia.

The following Tables, V., VI., and VII., have been constructed from the records of the London and Dublin Pathological Societies, and from my own register, respectively. They exhibit, in summary, the latest available contributions, in a clinical aspect, to the pathology of fatty disease of the heart; and, commencing from the date of publication of Dr. Quain's valuable statistics (May, 1850), they may be regarded as supplementary to his Tables, and as representing the present state of clinical knowledge of the subject in the United Kingdom.

\* *Contributions to Practical Medicine*, by James Begbie, 1862.

TABLE V.—FATTY HEART.

*Pathological Society of London (from May, 1850).*

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Bagshawe	M.	..	Tailor	Many years	Palpitation, faintings, pain in chest	..	Syncope by rupture of aneurism of left v. on left side	Atheromatous	Slightly atheromatous	Heart thinned; layer of fat on surface; rupture of aneurism of left v.	Fatty degeneration (slight)	Coronary arteries calcified, especially branch supplying seat of aneurism; kidneys granular.
Dr. Baly	F.	52	A lady	3 years	Temporary right hemiplegia, headache, and "visus dimidiatus"; weak and intermitting pulse	Weak action and sounds of heart	Rupture of left v. on posterior surface	Atheromatous and calcified	Mitral slightly thickened; aortic also thick and calcified at root	Left v. hypertrophied and dilated; much fat at base and in sulci	Fatty degeneration; greater in seat of rupture	Old blood-clot in brain, and degeneration of arteries at its base; degeneration of coronary arteries, and advanced degeneration and closure of branch leading to seat of degeneration.
Dr. Barker	M.	45	Mariner	A few weeks	Dyspnoea and palpitation; pulse 120, moderately full, & jerking; dropsy	Extensive precordial dullness, yet no impulse to be felt, and sounds very faint	Died suddenly after excitement (syncope?)	Atheromatous	Aortic much diseased	Left v. $\frac{3}{8}$ -in. thick; right v. dilated; heart 28 oz.	Early fatty degeneration	Aneurism of abdominal aorta, and granular degeneration of kidneys; degeneration of arteries of brain, and local softening of latter.
Dr. Barlow	F.	29	"Irregular"	..	..	..	Coma, from injury of head	Slightly atheromatous	..	..	Fatty degeneration (general)	
"	M.	—	Cabman	..	..	..	Asthenia	Dilated and atheromatous	..	Dilated generally	Granular degeneration; more extensive in left v.	Brain reduced in volume, tough, and in the state of "fibrous degeneration" of Sims and Cruveilhier; coronary arteries atheromatous; kidneys small and cystic; areus senilis; emphysema of right lung.
Dr. Beith	M.	79	..	Some years	Dyspnoea, vertigo, faintings, pain in chest	..	Rupture through septum of v. extending through anterior wall of right v.; both v. covered with fat to depth of one-half; lived six days after signs of rupture of septum	Celcarious	Healthy	Heart hypertrophied, 17 oz.	Fatty degeneration, early stage	Coronary artery leading to rupture calcified and plugged.



Dr. Bristowe	M. 35	..	5 months	Cough, debility, weak and intermittent pulse	Those of pleuro-pneumonia engaging lower half of left lung	Asthonia	Healthy	Healthy	Recent pericarditis with effusion; no adhesion; heart covered with shreds of lymph, and substance yellow, to depth of half of right v., and one line of left v.	Yellow portion in state of fatty degeneration; remainder healthy; the former shading into the latter part, which was very brittle	Left pleuro-pneumonia, and right ditto in less degree; liver congested.
"	M. 65	..	..	Embarrassed and desponding	..	Rupture of left v. posteriorly	Atheromatous	Mitral and aortic slightly atheromatous, but competent	Hypertrophied (16½ oz.); right v. loaded with fat	Slight fatty degeneration generally, but advanced in seat of rupture	Coronary arteries atheromatous; not more so in seat of rupture than elsewhere.
"	M. 11	.	6 weeks	Depression, general purpuric mottling, hæmorrhages; finally, loss of vision; dilated pupils, and "muscae;" pulse large, and quick	..	Asthonia	Healthy	Healthy	Normal as to size; mottled; yellow on interior of left v.	Advanced fatty degeneration	Brain remarkably white and anæmic, otherwise healthy.
"	M. 26	Clerk	6 months	Jaundice, vertigo, vomiting, debility; pulse quick, and weak	Action and sounds of heart reported normal	Asthonia	..	Healthy	Both v. hypertrophied, but not dilated; weight 13½ oz.	Fatty degeneration of internal layer of both v.	Liver fatty.
"	M. 10	..	1 month	Diphtheria	..	Asthonia	..	..	Heart small and contracted	Fatty degeneration, especially in patches on inner surface of right v.	Kidneys fatty.
Mr. Canton	M. 49	Gentleman	..	..	..	Syncope	..	..	Left v. hypertrophied, except at apex, where it was thinned, so as to consist of endo- and pericardium only; this portion was of the size of a plover's egg; general adhesion of pericardium	Fatty degeneration advanced in vicinity of aneurism only	

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Mr. Canton ..	M.	72	Spring-maker	3 years	Dyspnoea, and occasional pain in chest	..	Rupture of aneurism of aorta with pericard. Cardiac syncope	Atheromatous	Healthy	Large and flabby	Fatty degeneration	Coronary arteries atheromatous and calcified.
Dr. Chlormely	M.	48	County baronet	5 years	Attacks of angina pectoris; gouty.	..		Atheromatous (and also pulmonary artery)	Healthy (slight thickening at base)	Ventricles slightly dilated and streaked yellow on both surfaces	Fatty degeneration	Coronary arteries calcified; arcus senilis.
Dr. J. Cockle	M.	40	Soldier (ex.)	2 years	Pains in chest and arms occasionally; inability to lie on left side; cough	Dull first sound at apex, <i>sharp</i> second at base; then a double pulsating tumor to right of sternum, with double sound; first sound in this situation became "crumpling" shortly before death	Rupture of bilocular aneurism of aorta into pericardium	Dilated and atheromatous	Healthy	Left v. hypertrophied	Fatty degeneration	Adherent pericardium.
Mr. Coulson	F.	76	..	..	..	..	Rupture of left v. behind apex	..	..	Covered with fat to one line from interior	Fatty degeneration, (advanced)	Coronary arteries calcified.
"	M.	46	..	..	None preceding the syncope, of half an hour's duration, in which he died.	..	Syncope by rupture of aneurism of aorta into pericardium Syncope	Atheromatous	..	Loaded with fat	Fatty degeneration	
Dr. Crisp ..	M.	39	School-master	10 months	Pain in precordium extremely acute and continual for the last few weeks of life	..		Atheromatous and contracted	Aortic valves shrunk and contracted	Both v. dilated and thickened	Fatty degeneration	
Dr. T. W. Davis	F.	64	Nurse	8 months	General dropsy, dyspnoea, weak and irregular pulse; angina pectoris	Extensive precordial dulness; turbulent auscultation of heart; double basic murmur	Rupture of right v. anteriorly	Atheromatous	Aortic much disorganized	Right v. thinned & ruptured in front; left v. dilated and thick	Fatty degeneration	Coronary arteries calcified, especially right
Dr. Dickinson	M.	36	..	Few months	Antecedent peritonitis, and acute pains in feet	..	Exhaustion	Slightly atheromatous	..	Large and flabby	Fatty degeneration	Adhesion in peritoneum, and coagulation of blood in arteries of lower limbs.

Dr. Dickinson	M.	41	Butler	2 years	Extreme anæmia and emaciation from want	Double murmur at lower sternum	Exhaustion	..	Mitral and tricuspid rough	Heart large and flabby, with superficial layers of fat; fawn-coloured and mottled yellow	Extreme fatty degeneration	All organs anæmic; blood watery.
Dr. Fuller	F.	40	..	7 months	General malaise after tapping an ovarian cyst 4 years previously; jaundice and œdema of feet	..	Progressive asthenia	..	Normal	Apparently normal	Fatty degeneration (advanced)	Tubercle in apex of right lung, and collapse of adjacent part of it.
Dr. G. Gibb..	F.	45	..	..	..	..	Apoplexy	Dilated and atheromatous	Healthy	Left v. hypertrophied	Fatty degeneration of left v.	Liver fatty; arteries of brain atheromatous, and clot of blood projecting into right lateral ventricle.
"	M.	60	..	..	Those of phthisis	..	Asthenia	Atheromatous and calcareous	..	Heart normal in size, but soft	Fatty degeneration (advanced)	General calcification of arteries, including coronary; fatty liver and kidneys; tubercle and cavities in lungs; larynx and trachea calcified.
"	M.	46	..	..	..	..	Cerebral hæmorrhage	Atheromatous	..	Heart large	Fatty degeneration	Larynx and trachea calcified.
Dr. Greenhow	M.	65	Painter	8 years	Cough, and shortness of breath	Systolic murmur at lower sternum; extended precordial dulness, and weak impulse	Asthenia	Atheromatous	None	Heart large (25 oz.), and generally dilated	Fatty degeneration	Emphysema of lungs, and some cheesy tubercle.
Dr. Habershon	F.	23	"Dissolute"	..	Dyspnoea; articular rheumatism	Aortic-diastolic murmur; action of heart tumultuous	..	Asphyxia	Aortic valve disorganized	Heart slightly enlarged; small aneurism in wall of left v.	Fatty degeneration, in patches and streaks	Kidneys cystic; double pleuritis and pericarditis with effusion.
Dr. G. Harley	F.	65	..	A fortnight	Cough and slight dyspnoea	..	Rupture of left v. anteriorly	..	..	Normal as to size, but soft, pale, and flabby	Fatty degeneration	All organs anæmic.
"	F.	28	Householder	6 months	Spasmodic dyspnoea, weakness, and despondency	..	Syncope	..	..	Heart large, pale, flabby, and covered with fat	Fatty degeneration (advanced)	Liver fatty; spleen enlarged.
Dr. Hillier ..	M.	5	..	3 years	Enlarged lymphatic glands and spleen; convulsions before death	..	Exhaustion	..	..	Soft, pale, and mottled yellow	Fatty degeneration (extreme)	..
Mr. Holmes ..	M.	69	..	..	Suffering from stricture and stone; pulse weak and failing	..	Syncope	..	Healthy	Heart 14½ oz., pale and flabby	Fatty degeneration	..

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Handfield Jones	M.	62	..	..	None preceding final seizure	..	Syncope, after a journey of 86 miles	..	Mitral inadequate	Heart enlarged, 2½ oz.; left v. hypertrophied	General fatty degeneration	Ulcers of inner surface of stomach, regarded as result of fatty change.
Mr. A. Leggett	M.	18	Gentleman	An invalid from childhood	General paralysis	Sounds and impulse of heart reported healthy shortly before death	Right pneumonia	..	..	Normal	Fatty degeneration of voluntary muscles; softening of spinal cord; right basic pneumonia.	Fatty degeneration of voluntary muscles; softening of spinal cord; right basic pneumonia.
Dr. Leared ..	F.	11	..	6 months	General anemia, dyspnoea; vomiting; quick and weak pulse	A murmur with the first sound	Asthenia	..	Healthy	Somewhat large, pale, and flabby	Fatty degeneration	Pulmonary emphysema (slight), with hydro-thorax.
Dr. Lichtenberg	M.	29	..	Some time	Palpitation and dyspnoea	Double murmur at base	Rupture of aneurism of aorta into right ventricle	Atheromatous	Aortic valve thick and soft	Heart large, thin, and flabby; also yellow (superficial fat ?); aneurism, size of marble, projected into tricuspid orifice, and burst	Fatty degeneration	Liver and kidneys fatty.
Dr. J. W. Oglet	M.	69	Workman	..	Had two fits of apoplexy previously	..	Apoplexy	Atheromatous	..	..	Fatty degeneration, (early stage)	Atheroma of coronary and of cerebral arteries, with softening of brain and sanguineous effusion.
"	M.	54	..	..	Pain in back; dysphagia	..	Hæmoptysis by rupture of aneurism into cavity formed by pneumonia in base of right lung	Atheromatous	Healthy, but mitral orifice dilated	Loaded with fat; large and thinned	Fatty degeneration	Base of right lung excavated and communicating with aneurism of inferior thoracic aorta.
"	M.	Mid. age	..	..	Frequent fits, presumably syncope	..	Fit after full meal	..	..	Normal	Fatty degeneration	Aneurism of left middle cerebral artery which had burst into left lateral v.; pleuritic adhesion, and granular kidneys; meninges.
"	M.	39	Fishmonger	7 months	Ascites; slight jaundice	Basic-systolic murmur	Asthenia	..	Aortic valve ulcerated	Large and flabby	Fatty degeneration	Liver cirrhotic and fatty; kidneys fatty; spleen enlarged.
Mr. J. Part ..	F.	73	..	..	Those of enlarged liver and spleen	..	Exhaustion	..	..	Large and fatty	Fatty degeneration	Spleen greatly enlarged and fibrous; liver also greatly enlarged and fatty.



Dr. Peacock	M.	62	Gentleman	4 days	None preceding final illness	..	..	Aortic and mitral opaque and orifice of aorta reduced	Right v. covered with fat; left v. thick; heart enlarged (14½ oz.)	Fatty degeneration (general) greater in seat of rupture	Coronary arteries atheromatous and calcareous.
Dr. Pollock	F.	75	..	..	Pain in head for a fortnight before death	..	Found dead in bed, apparently from angina pectoris	..	..	Fatty degeneration	Arteries at base of brain calcified.
Dr. Quain	F.	59	A lady	7 years	Three attacks of apoplexy, and several of angina pectoris	Weak, dull, and prolonged first sound; sharp second sound	..	Healthy	Heart hypertrophied (14 oz.); right a. dilated and thick; right v. thick	Fatty degeneration & growth; former limited to part of septum and anterior wall of left v.	Coronary artery leading to degenerated part, calcified.
"	F.	68	A lady	2 years	Dyspnoea, oppression; numbness in left fingers and irregular pulse two days before death; fits of angina pectoris	..	Rupture of left v. on anterior surface	..	..	Local fatty degeneration in seat of rupture mixed with fibrous do.	Fatty liver, and calcification of coronary artery leading to degenerated part of left v.
"	F.	66	A cook	Some time	Shortness of breath on ascending heights, and slight pain in chest; weak, irregular, and intermittent pulse	Weak action and sounds, especially second	..	Healthy	Right v. dilated, thinned, and loaded with fat; left v. dilated only	Fatty degeneration, greater in seat of rupture	Coronary artery leading to seat of rupture calcified and obstructed.
"	F.	29	Prostitute	..	Phagedenic ulcer of pudenda	..	Asthenia or "cardiac syncope" under chloroform	Healthy	Dilated, flabby, and covered with fat	Fatty degeneration	All healthy.
"	F.	Aged	..	..	Cough and dyspnoea	..	Rupture of left v. anteriorly	..	Dilated, flabby, and covered with fat	Fatty degeneration greater near seat of rupture	Coronary artery leading to seat of rupture calcified and blocked.

TABLE V.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Quain ..	M.	58	Farmer	2 years	Attacks of syncope	..	Rupture of left v. anteriorly	..	Healthy	Heart slightly enlarged	Fatty degeneration; greater in seat of rupture	Coronary artery leading to seat of rupture calcified.
"	M.	73	Gentleman	8 years (?)	Left hemiplegia; Shortness of breath on making ascents; pain in chest nine days before death	..	Rupture of left v. anteriorly	..	..	Loaded with fat, and thinned	Fatty degeneration	Coronary arteries atheromatous.
Mr. R. R. Robinson	M.	43	Carpenter	6 months	Dyspnoea, faintness, and occasional palpitation; very weak pulse; enlarged liver; albuminuria; and latterly, orthopnoea; cedema, and gangrene of legs, and respirations only nine in the minute	Rapid and weak action of heart	Asthenia	Dilated and atheromatous	Aortic diseased	Both v. dilated and thick; pericardium adherent, and calcareous plate in false membrane; congenital opening in pericardium, through which left a. appendix was visible	Fatty degeneration	Liver "nutmeg" and fatty; kidneys fatty.
Dr. J. S. Ramskill	F.	45	A lady	Some time	General symptoms of fatty heart	..	Rupture of left v. anteriorly	Atheromatous	Aortic atheromatous at base	Heart small, soft, and covered with fat	Fatty degeneration	Right pleuro-pneumonia; coronary arteries calcified.
Dr. Thudichum	F.	33	..	8 years	Uterine and intestinal derangement	..	Right pleuro-pneumonia	Atheromatous	Aortic cribriform	Both v. dilated and thinned	Fatty degeneration and "green pigmental" ditto (?)	
Doctor Ogier Ward	F.	10	..	6 years	Chorea, pallor, lividity and dyspnoea; feeble, quick, and irregular pulse	Dulness and râles over bases of both lungs; extension of precordial dullness; murmur with first sound (presystolic?)	Asthenia	..	Endocardium of left a. opaque, and auricular surface of mitral valves "banded"; orifice narrowed (?)	..	Fatty degeneration (slight)	
Dr. Wilks ..	M.	61	Coal dealer	8 months	Attacks of angina pectoris	Sounds muffled	Fit of angina pectoris	..	Healthy	Left v. dilated; heart loaded with fat	Fatty degeneration	Coronary arteries calcified.
"	M.	61	Lunatic	..	..	..	Rupture of left v. anteriorly	..	..	Normal, but covered with fat nearly to entire depth	Fatty degeneration at seat of rupture (advanced)	Coronary arteries calcified, and branch leading to rupture plugged.

## SYNOPSIS OF TABLE V.

SEX :		Cases.
Males . . . . .		35
Females . . . . .		20
		—55
AGES :		
Under twenty years . . . . .		6
Twenty to thirty years . . . . .		6
Thirty to forty . . . . .		5
Forty to fifty . . . . .		11
Fifty to sixty . . . . .		4
Sixty to seventy . . . . .		13
Seventy to eighty . . . . .		6
Not stated . . . . .		2
"Middle aged" . . . . .		1
"Aged" . . . . .		1
		—55
DURATION OF ILLNESS :		
Minimum . . . . .	four days	
Maximum . . . . .	eight years	
SYMPTOMS :		
Faintness, intermittent and irregular pulse . . . . .		15
Paroxysmal dyspnoea . . . . .		16
Angina pectoris . . . . .		5
Dropsy . . . . .		5
Arcus senilis . . . . .		6(?)
Not stated . . . . .		8
		—55
PHYSICAL SIGNS :		
Aortic-systolic murmur . . . . .		4
Aortic-diastolic murmur . . . . .		1
Double aortic murmur . . . . .		3
Mitral-systolic murmur . . . . .		1
Left presystolic murmur . . . . .		1
Dull first sound . . . . .		5
Sharp second sound . . . . .		2
Not stated . . . . .		38
		—55
MODE OF DEATH :		
Asthenia . . . . .		23
Angina pectoris . . . . .		3
Asphyxia . . . . .		1
Coma . . . . .		3
Rupture of heart . . . . .		14
Rupture of aorta . . . . .		6
Not stated . . . . .		5
		—55
STATE OF AORTA :		
Atheromatous . . . . .		24
Not stated . . . . .		28
Healthy . . . . .		3
		—55
STATE OF VALVES :		
Normal . . . . .		16
Aortic affected . . . . .		9
Mitral affected . . . . .		6
Not stated . . . . .		24
		—55
STATE OF CAVITIES AND WALLS OF HEART :		
Fatty deposit . . . . .		12
Cavities thinned . . . . .		18
Cavities thickened . . . . .		13
Not stated . . . . .		12
		—55
STATE OF FIBRES :		
Advanced fatty degeneration . . . . .		50
Granular ditto . . . . .		5
		—55
STATE OF OTHER ORGANS :		
Fatty liver . . . . .		7
Fatty kidneys . . . . .		5
Granular kidneys . . . . .		2
Coronary arteries diseased . . . . .		21
Cirrhosis of liver . . . . .		1
Not stated . . . . .		19
		—55

TABLE VI.—FATTY HEART.  
*Dublin Pathological Society (from and including 1854-5).*

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Bennett.	F.	24	..	4 years	Dyspnoea; œdema	Occasional systolic murmur at right apex	..	Normal, but congested	Normal	Normal	Fatty degeneration in patches in patches	
Dr. H. Benson	M.	64	Gentleman	..	Dyspnoea on ascending steps; pains in arms and head; fainting	Weak action of heart	Syncope from rupture of left v. on posterior surface	Incipient atheroma	Normal	..	Advanced fatty degeneration	
Dr. Gordon	M.	41	..	..	Weak and intermittent pulse; cold and congested surface; cough	Bronchial râles	Angina pectoris	..	..	Thinned, and nearly converted into fat; i.e., loaded with fat	..	Congestion of bronchial membrane, & pulmonary apoplexy.
"	M.	14	Inmate of an orphanage	..	Weakness, drowsiness; quick, weak pulse; suddenly pain, and loss of heat sensation and motion in right lower limb, followed by gangrene of the toes; sore throat; dyspnoea; pain at precordium	Weak, but regular action of heart; sounds feeble, but normal; no murmur	Asphyxia slowly produced	Atheromatous	Normal	..	Fatty degeneration. ("granular")	Lungs emphysematous and fatty; liver and kidneys fatty; thrombosis of main artery of right lower limb, from common iliac downwards, with inflammation of its lining membrane.
Dr. Hayden.	M.	50	Blacksmith	11 months	Irregular, quick, and falling pulse; lividity and anasarca; albuminuria	Feeble action and sounds of heart	Asthenia	..	Normal	Dilated, hypertrophied and loaded with fat	Fatty degeneration	Kidneys fatty.
"	M.	60	Gunsmith	8 months	General dropsy, including genitals; irregular and intermittent pulse; sighing, and occasional irregularity of breathing; enlargement of liver, and jaundice; visible pulse; hæmoptysis; erysipelas and gangrene of right side	Increased area of precordial dulness; systolic murmur at apex, left axilla and back; diastolic murmur at base, and want of correspondence between radial pulse and action of heart	Asthenia	Dilated and atheromatous	Normal; aortic incompetent by dilatation of aorta	Right v. dilated; left v. dilated and thick; heart 25½ oz.	Fatty degeneration, advanced	Liver fatty; right lung hyperemic; aorta, carotids, right subclavian and brachial arteries inflamed.
"	M.	69	Cabinet-maker	..	Lividity, œdema, hæmoptysis; failing pulse, enlarged and rough liver; slow and curving	Rapid and irregular action of heart; musical systolic murmur at apex, and in left axilla	Coma	..	Normal, except a few spicula at base of nozzle	Right v. and a. dilated, and loaded with fat; left v. thickened; heart 18 oz.	Fatty degeneration	Liver enlarged, and cirrhotic; kidneys large and fatty.



Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden ..	M.	62	Architect	..	General neuralgic pains, and localized in right mammary region ; dry cough	Dull first, and sharp second sound	Rupture of aneurism into pericardium	Atheromatous and dilated into an aneurism	Normal	Left v. hypertrophied and dilated, and loaded with fat, as were also right a. and v. to greater depth ; a thin but healthy stratum of muscular fibre alone remaining on internal surface	Healthy	Aneurism at root of aorta.
Dr. Hewitt ..	F.	..	..	..	Dyspnoea only	None detected, as no cardiac disease was suspected	Rupture of left v. at base posteriorly	..	Normal	..	Fatty degeneration	..
Dr. Jennings	M.	40	..	..	Debility, fluttering and irregular pulse, dyspnoea	Weak and irregular action of heart	Asthenia	Atheromatous	Normal	Atrophied, thinned, and covered with fat	Fatty degeneration	Double pulmonary apoplexy.
Dr. H. Kennedy	M.	68	..	..	Bronchitis, oedema of feet, regular pulse	Impulse and sounds feeble	Suffocation	..	Normal	Thinned and enlarged, and loaded with fat, especially right v. ; adherent and ossified pericardium	Fatty degeneration	Bronchial congestion.
"	M.	65	..	Some mths.	Feeble pulse, lividity and anasarca ; dyspnoea	Extended precordial dulness	Suffocation	..	Normal	Thinned and loaded with fat ; acute pericarditis, with effusion	Fatty degeneration	..
Dr. Kidd ..	F.	23	..	..	Dyspnoea, dilated pupils, sinking	..	Asthenia	..	..	Thinned and almost entirely replaced by fat over right v. ; left v. pale	Fatty degeneration	Liver fatty.
Dr. Law ..	F.	67	..	..	Anasarca ; dyspnoea	Was in <i>extremis</i> , and no physical examination made	Asphyxia	..	..	Excentric hypertrophy of left v. ; thinning and dilatation of right v., walls of which were almost entirely replaced by fat	..	Lungs emphysematous.

TABLE VI.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. T. E. Little	M.	65	Gentleman	..	Corpulence; two attacks of oppression and vomiting a few hours before death	..	Syncope	..	Normal, except a few spots of atheroma, and same in coronary arteries	Heart enlarged and loaded with fat; both a. thinned; left v. ruptured on anterior surface near apex, and partially ruptured near the same point	Fatty degeneration, and interstitial deposit	
Dr. M'Dowel	F.	16	..	..	Fever, convalescence, suffocation, catarrh, and sinking	No note	Sinking and asphyxia	..	..	Heart small; right v. almost entirely converted into fat; left v. pale	Fatty degeneration	Bronchi congested; air and oil in blood.
Dr. Murney..	F.	60	Bonnet-maker	40 years	Recurrent palpitation; debility; anæmia; rapid pulse in the paroxysms; 70 ordinarily	Weak sounds; only the first audible in paroxysms; weak impulse; jugular pulsation	..	..	Normal	Thinned, and in advanced state of fatty degeneration	Fatty degeneration	General emphysema.
Dr. W. Moore	M.	55	Waiter	..	Lividity, dyspnoea, anasarca; faintings; irregular and intermittent pulse	Weak, irregular, and intermittent action of heart	..	..	..	Dilated; right v. nearly replaced by fat	Fatty degeneration	

## SYNOPSIS OF TABLE VI.

SEX :		Cases.
Males . . . . .		12
Females . . . . .		6
		—18
AGES :		
Under twenty years . . . . .		2
Twenty to thirty years . . . . .		2
Forty to fifty . . . . .		3
Fifty to sixty . . . . .		2
Sixty to seventy . . . . .		9
		—18
DURATION OF ILLNESS :		
Minimum . . . . .	eight months.	
Maximum . . . . .	forty years.	
SYMPTOMS :		
Faintness, intermittent and irregular pulse, etc., . . . . .	in 12	
Paroxysmal dyspnoea . . . . .	" 1	
Angina (pain, palpitation, and dyspnoea) . . . . .	" 2	
Dropsy . . . . .	" 8	
Arcus senilis . . . . .	" 1	
		—18
PHYSICAL SIGNS :		
Irregular action of heart . . . . .	3	
Intermittent ditto . . . . .	1	
Aortic-diastolic murmur . . . . .	1	
Mitral-systolic murmur . . . . .	3	
Dull first sound . . . . .	7	
Sharp second sound . . . . .	1	
Not stated . . . . .	2	
		—18
MODE OF DEATH :		
Asthenia . . . . .	5	
Angina pectoris . . . . .	1	
Asphyxia . . . . .	4	
Coma . . . . .	1	
Rupture of heart . . . . .	2	
Rupture of aorta . . . . .	1	
Not stated . . . . .	4	
		—18
STATE OF AORTA :		
Atheromatous . . . . .	5	
Congested . . . . .	1	
Not stated . . . . .	12	
		—18
STATE OF VALVES :		
Normal . . . . .	12	
Aortic affected . . . . .	1	
Not stated . . . . .	5	
		—18
STATE OF CAVITIES AND WALLS OF HEART :		
Fatty deposit, . . . . .	in 12	
Cavities thinned . . . . .	" 10	
Cavities thickened . . . . .	" 4	
		—18
STATE OF FIBRES :		
Advanced fatty degeneration . . . . .	14	
Granular ditto . . . . .	1	
Healthy . . . . .	1	
Not stated . . . . .	2	
		—18
STATE OF OTHER ORGANS :		
Pulmonary apoplexy . . . . .	1	
Fatty liver . . . . .	3	
Fatty kidneys . . . . .	2	
Cirrhosis of liver . . . . .	1	
Not stated . . . . .	11	
		—18

TABLE VII.  
Author's Cases.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden..	M.	69	Float driver	..	Chronic cough; orthopnoea; recurrent vertigo; pulse 108, regular; rigidity and tortuosity of temporal arteries; urine, sp. gr. 1.025, with a trace of albumen; hæmoptysis; jugular pulsation; cedema; erysipelas of genitalia, and finally somnolence, and contraction of the pupils	Diffused and feeble cardiac impulse; no apex-pulsation to be felt, a murmur replacing first sound at apex and sound at apex and to left; transmitted systolic murmur at base; weak second sound; signs of pulmonary emphysema	Asthenia	Aorta near ventricle was greatly dilated and adherent to the pericardium in patches; the orifice was not dilated, and the valves were competent	..	Heart enlarged, soft, and flabby; it weighed 23 ozs.; right v. covered by a layer of fat two lines thick; an irregular white patch on its surface; right a. and v. dilated, and latter likewise thinned; tricuspid opening dilated, and valves incompetent; right chambers contained a yellow thrombus, which passed through tricuspid opening, and bound up antero-left segment of valve so as to render it incompetent; left a. and v. dilated and thickened; all the valves structurally sound, mitral valves apparently competent	Granular and fatty degeneration and obsolescence of deeper fibres of both v., and granular change only of superficial fibres	Liver, spleen, and lungs congested.
"	F.	45	..	Some years	Cough, dyspnoea, debility, weak and intermittent pulse; hæmoptysis	Sharp first sound and accompanying soft murmur at apex	Asthenia	..	Mitral tuberculated	Heart large and covered with fat; right v. dilated	Granular stage of fatty degeneration	Pulmonary emphysema and lobular interstitial pneumonia.
"	M.	68	Soft-goods dealer	11 weeks	Faintings, cedema, quick and irregular pulse; hæmoptysis	Feeble impulse; systolic murmur at left apex; ditto at lower sternum and in ascending aorta; faint second sound; rough systolic murmur (pericardial) over root of pulmonary artery	Asthenia	Atheromatous	Mitral and aortic thick, and the latter calcareous	Heart large and covered with fat; left v. concentrically hypertrophied	Fatty degeneration(?)	Lungs (apices of both) enveloped in false membrane, and right lung congested.
"	M.	66	A porter	12 months	Dyspnoea; cedema; quick and irregular pulse, and venous pulsation in the neck; and a few days before death, left hemiplegia and inability to articulate	Quick and irregular action of heart; apex displaced to left, and first sound abnormally clear	Exhaustion	..	All the valves normal	Heart large and flabby, with slight deposit of fat; right v. dilated and thin	Advanced fatty degeneration	Blood clot in right anterior lobe of cerebrum, and fibrinous plug in right middle cerebral artery; granular degeneration of the kidneys.



TABLE VII.—Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden..	M.	40	Reduced gentleman	Some weeks	Derangement of stomach and "anomalous nervous symptoms"	..	Syncope	Dilated and atheromatous, and lining membrane fissured	All sound, except one segment of mitral, which was slightly thickened	Heart large (18½ ounces) and covered with fat; left v. dilated and thickened	Advanced fatty degeneration, especially of some of the fleshy columns of left v., which were mottled yellow	Lungs congested.
"	M.	70	Tailor	6 years	Dyspnoea; general dropsy; arcus senilis; cough and frothy expectoration; irregular breathing; feeble pulse; rigidity of arteries; throbbing of carotids	Feeble cardiac impulse; systolic murmur at lower sternum; sharp second sound	Syncope	Dilated and atheromatous	Aortic thickened at root; one of mitral opaque			
"	M.	62	Carpenter	12 months	Paroxysmal dyspnoea; "ascending and descending" respiration; failure of pulse; cedema and paralysis of left arm	Weak and rapid action of the heart; clear sounds at lower sternum, and systolic murmur	Asthenia	Greatly dilated, and slightly atheromatous	Mitral and aortic valves thickened and inadequate	Right chambers normal; left v. thickened, except apex, where it was thinned	Fatty degeneration of left v.	Serous effusion into right pleura; other organs not examined.
"	M.	60	Labourer	3 months	Dyspnoea; dizziness; "ascending and descending" respiration; no cedema; somnolence	Rapid and irregular action of the heart; double murmur at base, and systolic only in the carotids; diastolic murmur, and a quasi-presystolic murmur at the apex	Asthenia	Dilated and calcareous	Aortic thick & incompetent; warty vegetations at root of mitral, which were otherwise healthy and competent	Heart enlarged (19 ozs.); left v. thickened	Fatty degeneration, advanced in right, & slight in left v.	
"	M.	34	..	..	Delirium tremens; rapid pulse; sleeplessness	No perceptible cardiac impulse; sounds obscure; no murmur	Syncope	..	All healthy	Heart enlarged (19½ ozs.); left v. dilated; heart covered with fat	Granular degeneration of internal layers of left ventricle "Glassy" stage of fatty degeneration	Lungs congested.
"	F.	40	..	..	Acute articular rheumatism; great depression and weak pulse	..	Syncope	..	All healthy	Normal as to size, etc.		

TABLE VII. — Continued.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Cavities and Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden.	F.	28	Householder	16 years	General dropsy; dyspnoea; lividity; albuminuria; venous turgescence in neck	Extended precordial dulness; strong impulse behind lower sternum; systolic bellows-murmur at apex, and at base; second sound dull in aorta, but sharp in pulmonary artery	Coma	Narrowed, and of a crimson tint internally	Mitral valves agglutinated and leaving only a narrow slit; aortic opaque & slightly rough, but competent	Heart enlarged (13½ ozs.); right chambers dilated and thickened; left pulmonary veins dilated, and a. thickened; left v. thickened; tricuspid and pulmonary orifices dilated	Granular degeneration and loss of transverse striation	Much liquid in the serous cavities of chest and abdomen; congestion and slight emphysema of lungs; kidneys thick and yellow in cortex.
"	M.	53	Gardener	3 weeks	Recurrent syncope; cough; dyspnoea, and irregularity of breathing	Systolic murmur at lower sternum, and absence of second sound here	Syncope	Greatly dilated, rough, and fissured	Aortic slightly thick and opaque, but competent	Heart enlarged (18 ozs.) and fatty on surface	Fatty degeneration and partial obsolescence of fibres	Lungs emphysematous and congested, and traces of former right pleuritis; dilatation of trachea.
"	F.	48	"	Some time	Swelling of feet; weak and visible pulse; convulsive seizure	Apex-beat displaced to left; systolic apex and basic murmur, latter audible in the carotids	Asphyxia after convulsions	Healthy	Anterior segment of mitral thickened, and a flake of fibrin on its ventricular surface which floated in to orifice of aorta	Hypertrophy and dilatation of left v.; deposit of fat on surface of right v. which was dilated and thin, as was also right a.	Granular degeneration of the heart	Serous effusion into pleure and pericardium.
"	M.	45	House-painter	9 months	Sudden hemiplegia; epistaxis; vomiting; shortness of breath; pallor; albuminuria, & granular tube-casts; erysipelas; uræmic coma; rapid pulse; rigidity of arteries; alternate dyspnoea and apnoea	Displacement of apex to left; apex systolic murmur; second sound sharp at apex	Coma	Dilated and atheromatous	Fibrinous deposit upon the mitral valve	Hypertrophy of the left v.; heart weighed 23½ ozs.	Granular degeneration of the heart	Cirrhosis of liver, kidneys, and spleen.
"	F.	18	Orphan child of reduced gentleman	Some time	Anæmia and great debility; dropsy; uricælow in sp.gr., and containing hyaline casts;	No cardiac impulse; faint sounds	Asphyxia	Normal	Healthy	Hypertrophy with dilatation of left v.; dilatation of right v.; heart weighed 15½ ozs.	Granular degeneration of both v.	Granular degeneration of the kidneys; enlargement and congestion of liver.

Author.	Sex.	Age.	Occupation.	Duration of Illness.	Symptoms.	Physical Signs.	Mode of Death.	State of Aorta.	State of Valves.	State of Walls of Heart.	State of Fibres.	State of other Organs.
Dr. Hayden..	M.	45	Engineer	2 years	Recurrent vertigo; hæmoptysis; paroxysmal dyspnoea; lividity	Diastolic impulse; displacement of apex outwards and downwards; faint first sound; post-diastolic basic murmur	Syncope	Dilated and atheromatous	Onesegment of aortic slightly thick, but competent	Enlargement of heart (23½ ozs.); hypertrophy and dilatation of both v., and thrombosis	Granular degeneration	Cirrhosis of liver.
"	M.	48	Labourer	3 years	Irregular and suspicious breathing; weak and intermittent pulse; albuminuria and polyuria; œdema of feet; vomiting, and diarrhoea	Displacement of apex to left; sounds sharp and clear; doubling of first sound at base	Coma	Dilated, and its lining membrane injected	Normal	Enlarged (14½ ozs.), and a thick layer of fat on base and on right v.	Healthy	Granular degeneration of kidneys.
"	M.	56	Farmer	17 months	Angina pectoris; recurrent feeling of faintness; œdema of feet and legs; orthopnoea	Extended precordial dulness; strong impulse; dull first sound; basic diastolic murmur	Exhaustion	Dilated and atheromatous	Normal	Enlarged and flabby (27 ozs.); large deposit of fat on surface; dilatation of right chambers; thickening of left ventricle	Fatty degeneration	Hydro-pericardium.
"	M.	44	Coachman	2 months	General dropsy; albuminuria; jaundice, erysipelas, and gangrene; dyspnoea; weak pulse; irregular breathing; hæmoptysis	Labourer action of heart	Asthenia	Healthy	Normal	Enlarged (14½ ozs.); right chambers soft and thin; left chambers dilated and thickened	Fatty degeneration	Dropsy of serous cavities; fatty change of kidneys; fatty liver and spleen; pulmonary apoplexy.
"	M.	44	Discharged soldier	4 months	General dropsy; dyspnoea; quick pulse; albuminuria; orthopnoea; hæmatemesis	Extended precordial dulness, and strong cardiac impulse	Syncope	Dilated	Normal	Enlarged and loaded with fat; right cavities dilated, and left v. concentrically; hypertrophied	Granular degeneration	Cirrhosis of liver, and fatty disease of kidneys; hydro-pericardium.
"	M.	43	Pedlar	6 months	Abdominal pain; irregularity of bowels; emaciation; tumor in hypogastrium	Limited area of precordial dulness; weak action of heart; weak sounds	Exhaustion	Reduced in size	Normal	Atrophied; (6 ozs.)	Granular degeneration	Adhesion and calcification of the pericardium.
"	M.	56	Labourer	26 months	Vertigo; faintness; dyspnoea; visible pulsation at wrists; cough and expectoration	Feeble and diffused impulse of heart; systolic basic murmur; sharp and clear second sound, which was afterwards entirely suppressed; double precordial <i>frottement</i>	Asthenia	Dilated and atheromatous	Aortic valve and base of one segment of mitral v. calcified	Enlargement of heart (23½ oz., with pericardium attached); surface rough of lymph; hypertrophy and dilatation of both ventricles	Granular degeneration	Lungs emphysematous, congested, and enveloped in false membrane, by which they were universally adherent to walls of chest.

## SYNOPSIS OF TABLE VII.

SEX:*							Cases.
Males	.	.	.	.	.	.	17
Females	.	.	.	.	.	.	5
							—22
AGE:							
Under twenty years	.	.	.	.	.	.	1
Twenty to thirty years	.	.	.	.	.	.	1
Thirty to forty	.	.	.	.	.	.	1
Forty to fifty	.	.	.	.	.	.	11
Fifty to sixty	.	.	.	.	.	.	2
Sixty to seventy	.	.	.	.	.	.	5
Seventy to eighty	.	.	.	.	.	.	1
							—22
DURATION OF ILLNESS:							
Minimum	.	.	.	.	.	three weeks.	
Maximum	.	.	.	.	.	sixteen years.	
SYMPTOMS:							
Faintness, intermittent or irregular pulse,	.	.	.	.	.	in 16	
Paroxysmal dyspnoea	.	.	.	.	.	" 11	
Angina pectoris (pain, palpitation, and dyspnoea)	.	.	.	.	.	" 1	
Dropsy	.	.	.	.	.	" 12	
Arcus senilis (noted)	.	.	.	.	.	" 1	
							—22
PHYSICAL SIGNS:							
Irregular action of heart,	.	.	.	.	.	in 2	
Aortic-systolic murmur	.	.	.	.	.	" 8	
Aortic-diastolic murmur	.	.	.	.	.	" 2	
Double aortic murmur	.	.	.	.	.	" 2	
Mitral-systolic murmur	.	.	.	.	.	" 7	
Left presystolic murmur	.	.	.	.	.	" 1(?)	
Dull first sound	.	.	.	.	.	" 4	
Sharp second sound	.	.	.	.	.	" 4	
							—22
MODE OF DEATH:							
Asthenia	.	.	.	.	.	17	
Asphyxia	.	.	.	.	.	2	
Coma	.	.	.	.	.	3	
							—22
STATE OF AORTA:							
Atheromatous	.	.	.	.	.	11	
Congested	.	.	.	.	.	2	
Healthy	.	.	.	.	.	3	
Reduced in calibre	.	.	.	.	.	1	
Not stated	.	.	.	.	.	5	
							—22
STATE OF VALVES:							
Normal,	.	.	.	.	.	in 9	
Aortic affected	.	.	.	.	.	" 8	
Mitral affected	.	.	.	.	.	" 1	
							—22
STATE OF CAVITIES AND WALLS OF HEART:							
Fatty deposit,	.	.	.	.	.	in 11	
Walls of chambers thinned	.	.	.	.	.	" 9	
Walls of chambers thickened	.	.	.	.	.	" 14	
							—22
STATE OF FIBRES:							
Advanced fatty degeneration	.	.	.	.	.	10	
Granular stage	.	.	.	.	.	11	
Healthy	.	.	.	.	.	1	
							—22
STATE OF OTHER ORGANS:							
Fatty liver	.	.	.	.	.	2	
Fatty kidneys	.	.	.	.	.	4	
Granular degeneration of kidneys	.	.	.	.	.	5	
Cirrhosis of Liver	.	.	.	.	.	3	
Healthy.	.	.	.	.	.	8	
							—22

\* There was no female over forty-eight years of age, and no male under thirty-four years of age.



The *treatment* of fatty accumulation upon the heart, when this condition can be inferred rather than positively diagnosed, differs in no respect from that of general obesity. The measures taken may be moderately active, if the person under treatment be young and adjudged structurally sound. Thus, if the pulse be strong and regular; the heart, as judged by percussion and auscultation, free from hypertrophy, dilatation, and valvular lesion; the aorta healthy, as determined by the quality of the cardiac sounds in the ascending aorta, and the character of the respiration; and the kidneys free from disease, as shown by a careful general and microscopical examination of the urine, under such circumstances, active exercise may be enjoined, especially that which quickens respiration and consumes heat-producing tissue; such as quick walking up hill, the use of the dumb-bells or quoits, racket-playing, etc. At the same time the use of hydrocarbons in food and drink must be restricted, especially sugar, fats, milk, and malt liquors. For the same reason rum and liqueurs ought to be forbidden. Wholesome nitrogenous food should be taken frequently to anticipate a craving appetite, but always short of satiety. A bath, either cold or tepid, should be frequently taken; the bowels should be well moved at least once daily, and the period assigned to sleep should not exceed seven hours out of the twenty-four. Alkaline waters, natural and artificial, such as those of Vichy and Kissingen, Seltzer, etc., may be freely drunk. This plan, by which the adipose tissue is *gradually* reduced, and then kept within moderate limits, is very different from the extreme and heroic system of Banting, by which it was proposed to *rapidly* dissipate fatty accumulations.

The system of Banting involved no little danger to the integrity of structures and organs, especially those subject to periodic and frequent distention, such as the heart and arteries, by the attenuation and weakening of their walls which necessarily follow the rapid removal of interstitial and superficial fat. There is, moreover, considerable danger of actual degeneration of tissue from abrupt reduction of the wonted supply of pabulum. Hence, Bantingism should, under all circumstances, be discontinued, even in the young and healthy. The plan thus briefly sketched should be put in operation in all cases, but modified according to age and

other circumstances, where there is a tendency to adipose development. Thus, in middle age, especially where there is such evidence of cardiac weakness as that afforded by feeble impulse and radial pulse, and palpitation and dyspnoea after a moderate walk, pedestrian exercise should be limited, but regular, and always, if possible, on level ground, in the open air, and short of fatigue. Even in such cases, if there be not conclusive evidence of structural change and advanced attenuation of the heart, moderate and regular exercise on foot, such as walking or playing billiards, should be recommended, as conducing not only to the reduction and limitation of fat, but to the healthy nutrition of the tissues of the body generally. I have already stated as my opinion, and I would repeat it here, that of the rare and peculiar condition of the heart in which its proper structure is represented, but in different proportions in the several cavities, by only a thin layer of muscle internally, the greater portion of the substance of its walls having been replaced by an ingrowth of fat from the outer surface, the thin residuum of muscle being, however, *structurally* sound, there is absolutely no positive evidence, general or physical (see "Aneurism," Mr. B.).

It would seem that in such cases, provided always the duty imposed upon the heart by exercise or excitement be not even *once* in excess of the ordinary routine, that the attenuated muscular wall is enabled by the soundness of its structure, and by the mechanical support which it receives on the outside from a thick and solid layer of adeps to perform its function without faltering. Where this condition is suspected, from the existence of a feeble but regular pulse, and the absence of precordial impulse conjoined with general obesity, positive diagnosis being, in the present state of knowledge, impracticable, physical effort and excitement of every kind should be strictly prohibited. Measures should be taken to improve the general nutrition in the hope of advancing that of the heart; and tonics, such as iron, quinine, and strychnia, should be given with a view to strengthening the portion of muscular substance which remains.

Where fatty degeneration of the heart actually exists, there cannot be a hope of equal benefit from treatment. Tissue is now in process of retrograde metamorphosis, and the utmost that can be hoped from treatment is to stay that fatal process, whilst

guarding against accidents from the changes which it has already wrought. Digestion should be promoted, the appetite, if possible, improved, and the bowels, which are usually confined, should be regulated by means of mild saline aperients. Medicines capable, at the same time, of improving the appetite and promoting the nutrition of the muscles by imparting tone and vigour to their action, should be steadily and perseveringly administered. Of these, the best are quinia and strychnia, given in moderate doses, and combined with iron where the anæmic aspect is presented. I usually direct the following: *R.* Sulph. quiniæ, grs. xxiv; Acid. sulphur. dil., ℥xxx; Liquor. strychniæ (B. P.), ℥xlviii; Sulph. ferri granulat., grs. viii; Syrupi flor. aurant., ʒiv; Aquæ font., ad ʒviij. A tablespoonful to be taken thrice daily.

Should there be evidence of sanguineous engorgement of the liver and of fatty accumulation in its cells, as furnished by enlargement and tenderness of that organ, and a jaundiced tint of the skin and conjunctivæ, then an occasional dose of blue pill, combined or not with resin of podophyllin according to the state of the bowels, should be directed. In the treatment of dropsy dependent upon fatty degeneration of the heart, a combination usually consecutive to chronic disease of the kidneys, reliance must be placed mainly upon active purgation with saline aperients, and the dry air-bath. Where the kidneys are merely congested, or secondarily engaged, the urine exhibiting slight opalescence when heated or tested with nitric acid, the specific gravity being 1·020 or upwards, after dry cupping and warm poultices to the loins, diuretics should be given. Of the latter, decidedly the best is digitalis; because it is not only the most efficacious as such, but also because it is the most active and reliable tonic stimulant of the heart which we possess. It is still more decidedly indicated if, in addition to the presence of dropsical effusion, the action of the heart be faltering and irregular. The effect of this medicine in restoring regularity of action to the heart, and increasing its contractile vigour and force, entitles it to the first place as a cardiac tonic. I prefer the tincture to all other preparations of this drug, and usually prescribe it as follows: *R.* Tinct. digitalis, ℥lxxx; Spirit. æther. nitros., ʒij; Aquæ font., ad ʒviij. An ounce every third hour. It is in cases of the former kind, namely, primary disease of the kidneys, fol-



lowed in succession by hypertrophy and fatty degeneration of the heart with atheromatous change of the arteries, that the most formidable of the complications of fatty disease of the heart, gangrene of the lower extremities, is most frequently met with. There is previously considerable oedema of the feet and legs with tension of the skin; an erysipelatous blush then appears upon the dorsum of one or both feet, or above the ankles, attended with elevation of temperature, and here one or more bullæ filled with amber-coloured serum, are formed; the surrounding surface is now congested, the temperature falls, and gangrene rapidly spreads from the bullæ as from so many centres. I have reason to believe that in such cases there is always the antecedent change of aortic arteritis which is intimately associated with the immediate and determining cause of gangrene; but, inasmuch as acute inflammation of the lining membrane of the aorta is always a sequela of atheromatous conversion of its walls, and as the latter is usually accompanied by dilatation of the vessel, we might expect to find rhythmical dyspnoea, or irregularity of breathing, a prominent feature of those cases in which gangrene of the lower limbs is a final symptom; and so it is. In all the examples of this truly formidable complication which have come under my notice, there were present rhythmical dyspnoea, and the sharp, clicking, second sound in the ascending aorta, indicative of atheromatous conversion of its walls. The gangrene is, I believe, immediately dependent upon local arteritis and thrombosis. If tension of the integument be excessive, acupuncture may be practised, or the skin, where most on the stretch, may be punctured freely with the point of a lancet at distant intervals; the seat of most active inflammation being, however, avoided. A lotion, consisting of 3ij of Goulard's solution to 3x of water, should be kept constantly applied to the inflamed portion of the skin, whilst those parts which are already livid, and in which the temperature has fallen below the normal standard, should be wrapped in cotton wadding, or soft wool.

In the treatment of the distressing form of dyspnoea, designated "of ascending and descending rhythm," I have found the inhalation of nitrite of amyl the most efficacious means of relief. Ten or twelve drops may be received into the folds of a pocket-handkerchief or napkin, and freely inhaled by the patient; or a



few whiffs may be taken, at the acme of respiratory distress, directly from a bottle containing the nitrite. The effect has been to mitigate the feeling of want of air, and to slow the respiration. The peculiar effect of this agent is to relax the systemic capillaries and increase the volume of blood circulating in the tissues. It was a knowledge of this action of the nitrite of amyl, that first suggested to me a trial of it for the relief of the distressing symptom under notice, which depends, as already shown, upon failure of tissue-respiration.

The treatment of the closely allied symptom of angina pectoris, so often presented in connexion with hypertrophy and fatty disease of the heart with atheroma of the arteries, especially in gouty subjects, should be conducted on the same principle; namely, that of promoting capillary circulation, and relieving the distended heart. For the accomplishment of this twofold purpose, I am not acquainted with any measures more efficacious than dry cupping over the chest, warm stimulating applications to the feet and legs, such as hot mustard baths or sinapisms, and nitrite of amyl given by inhalation, as described in the preceding paragraph. Dr. Strange, of Worcester, has found the hydrate of chloral, in doses of grs. xxv-xxx, most efficacious in the treatment of angina pectoris. It should be given in the above mentioned dose every hour, till relief, which usually follows the first dose, is obtained. Two cases are mentioned by Dr. Strange in support of his statement, and in one of these, a dose of chloral was given nightly for four months, with a few omissions; on these latter occasions only the angina returned.\* I cannot speak from personal experience of the treatment of angina with hydrate of chloral, but I would not hesitate to make trial of it, as recommended by Dr. Strange. The intercurrent angina of fatty heart is frequently dependent upon sub-acute aortitis; such cases are characterized by a feeling of great constriction at the upper portion of the sternum, and are best treated by leeching in this situation, and subsequently by the administration of small and repeated doses of grey, and antimonial powder.

A six weeks' residence at Kissingen or Harrogate, and a course of mild alkaline chalybeate waters, would be eminently condu-

\* *Medical Times and Gazette*, September 24th, 1870.

cive to the restoration of tone and vigour to those fibres of the heart which are suffering from feeble nutrition, but not yet actually in process of fatty change. Such fibres, which are characterized by granular dotting at intervals, transverse striation being still manifest over the greater portion of their length, may be found in greater or less number even in hearts most advanced in fatty metamorphosis. They are the representatives of healthy structure in the organ, and the sole agents in carrying on the circulation. Hence the importance of arresting the further process of change in them by promoting their nutrition.

I now proceed to submit examples of the condition discussed at such great length in the preceding pages.

CASE XLII.—*Pulmonary Emphysema and Interstitial Pneumonia; Mitral Regurgitation; Death. Fatty Accumulation upon the Heart, and Granular Degeneration of its Substance.*

Susanna H., aged forty-five years, was admitted into the Mater Misericordiæ Hospital, January 20th, 1865, suffering from cough with great dyspnœa and debility. Pulse 114, very weak, and intermittent. The cervical veins were distended, and there was venous and arterial pulsation in the neck. At the apex of the heart the first sound was clear and accompanied by a soft systolic murmur. Alcoholic stimulants were freely administered, and under their use her general condition was improved.

On the 14th of February, whilst the breathing was less embarrassed, the pulse was scarcely to be felt, and on the 15th streaks of florid blood appeared in the sputa; an event which, as she then admitted, had frequently occurred before. The apex-murmur, which up to this date continued to be audible, was not to be heard. During the two days immediately preceding her death, which took place on the 20th, no pulse could be felt at the wrist.

The body was examined on the following day. Both lungs were partially emphysematous, and several patches of interstitial pneumonia, in its early stage, were found in them. The bronchial membrane was congested, and the bronchial tubes contained much viscid mucus. There were several ounces of serum in the sac of the pericardium, and the heart, especially at its base, was

covered with a thick layer of fat. The right chambers were dilated and filled with dark coagulated blood. The left ventricle was somewhat thickened, but not dilated, and the mitral valve was tuberculated upon the edge. The aortic orifice was slightly contracted, but the valves were healthy and competent. No striæ were visible in the muscular fibres of the heart examined microscopically. The fibres were pale, and exhibited lines of refractile granules. To this case is appended in my register the following note, which I copy *verbatim*: "This with some other similar cases, viz., of partial emphysema with congestive bronchitis, suggests the reflection, that in such the aggravated dyspnœa, failure of circulation, and anasarca, are due, not so much to the affection of the lungs, as to fatty and weak heart, which I have always found to be an associated condition. Hence the benefit derived from the free use of brandy and other stimulants." In such cases, likewise, mitral reflux, not dependent upon valvular disease, is of frequent occurrence, from weakness and yielding of the walls of the left ventricle at the acme of systole.

CASE XLIII.—*Aneurism of the Ascending Portion of the Arch of the Aorta; Inadequacy of the Aortic Valves; Hypertrophy and Fatty Degeneration of the Heart; Death.*

Rev. Mr. R., aged forty-five years, was received into the Mater Misericordiæ Hospital as a private patient on the 2nd March, 1865. He was a large man, and had been very corpulent. Had irregular and intermitting pulse for the last ten years, and was told that he had fatty disease of the heart.

In July, 1864, he had one or two attacks of vertigo, and in November of the same year he caught cold, and had a bad attack of bronchitis; his breathing then became short for the first time.

On the night of January 15th, 1865, when about retiring to bed, he felt a sensation of tightness about the upper part of the sternum, accompanied with hurried and difficult respiration, and with great excitement and alarm; he did not venture to go to bed that night, and he has not been since able to lie down. The feet began to swell about the 15th February.

When admitted, he was remarkably pale, and rather thin; the pupils were much contracted, even in the shade; the figure was bent, and there was complete orthopnoea. He experienced most relief by leaning forward in the sitting posture. The lower limbs were throughout œdematous, and pitted on pressure, but were free from congestion. Urine loaded with lithate of ammonia, sp. gr. 1.027, and passed in small quantity; only twenty-two ounces in the twenty-four hours; it contained a trace of albumen. Bowels rather confined; pulse 90 to 96, full, soft, irregular, and occasionally intermitting, but not "late" in relation to the ventricular contraction. Cardiac impulse feeble; first sound ill pronounced. A sharp, whizzing murmur replaced the second sound at midsternum, was traceable upwards and to the right with increasing loudness, and attained its greatest intensity at the upper part of the sternum; in this situation there was a considerable circumscribed prominence engaging the right side of the sternum, and three superior costo-sternal articulations of the same side. Over this prominence, which was comparatively dull on percussion, the diastolic murmur, as already stated, was loudest; it extended into the right subclavicular region, but not into the left. Two sounds were heard over this prominence; viz., one corresponding to the first sound of the heart and similar to it in character, the second being the diastolic murmur, heard in its greatest intensity. The most careful examination failed to detect impulse over the prominence, or elsewhere, save in the normal situation of the heart. Neither impulse nor murmur was perceptible in the back. Respiration 30, feeble on right side, loud on left, and accompanied with coarse râles over both bases posteriorly. No visible pulsation of the arteries, and no thoracic pain, but a feeling of "tightness" behind the fourchette of the sternum. No distention of the superficial veins of the chest, but the right external jugular was distended at its junction with the subclavian. No difference between the radial pulse on the opposite sides. Diagnosis: Aneurism of the upper portion of the ascending aorta pressing on the right bronchus; this aneurism is probably false, one or more of the coats of the vessel having given way on the night of January 15th; aortic valves inadequate, most probably by simple dilatation of the orifice; and finally, hypertrophy of the heart from antecedent



atheroma of the aorta; fatty degeneration of the heart, in a not advanced stage; and engorgement of the liver.

On the 9th March a feeble impulse was felt over the sternal prominence at the end of expiration, the patient having on the preceding night experienced several attacks of paroxysmal dyspnoea. These attacks were readily brought on by mental excitement or physical exertion of any kind, and were promptly relieved by the application of a few leeches. He was usually incoherent at night, but collected during the day. Œdema extending upwards. Under the use of tincture of digitalis in doses of ℞, with an equal quantity of spirit of nitrous ether, and of elaterium and blue pill in small doses, the urine was increased in quantity, the œdema was somewhat reduced, and the pulse-rate brought down to 66-72.

On the 22nd March the pulse was very irregular; there were 14 beats within the first recorded period of ten seconds, 17 in the second, and 15 in the third corresponding period. He had a paroxysm of dyspnoea, during which it was thought he would have died; it came on without any assignable cause, was accompanied by dry, hacking cough, and lasted two hours.

On the 23rd, œdema had extended to the abdomen, which contained some liquid, and fine crepitation was audible over the base of both lungs. There was likewise persistent dry cough. Infusion of digitalis was now given, in half ounce doses, with spirit of juniper and chloric ether; this produced copious diuresis, and was followed by some relief from the dyspnoea. The legs were subsequently punctured to avert gangrene of the over-stretched skin; on the 3rd April he spat some blood.

April 5th. Pulse 108, full and bounding. The urine contained a larger proportion of albumen, and the right foot, and lower portion of right leg, were tender and erythematous; œdema much reduced; heart's action strong; both sounds audible, but without murmur; two sounds were likewise audible over the sternal prominence, *but no murmur*; no dysphagia.

On the 6th, the murmur was again audible at the base of the heart, and over the sternal prominence, as before.

On the 14th, an eruption of the character of discrete variola appeared on the lower half of the right arm, and about the

elbow, but was strictly confined to these situations; the contents became opaque, and the pustules began to fade on the following day; and then, for the first time, there was visible pulsation in the radial arteries and carotids.

26th. The scrotum, which was greatly distended with serum, was punctured to-day. Respiration 36, performed rapidly, and at long intervals; great cardiac distress, and murmur nowhere audible; respiratory murmur very feeble over right front; loud over left.

He died on the 3rd May, and permission to examine the body could not be obtained. I think, however, the diagnosis made was fully warranted by the symptoms and physical signs exhibited. As the case is so full in detail, and in many respects so typical of its kind, I have deemed it worthy of publication, even without the proof afforded by dissection.

In connexion with the preceding case, the next, which in many respects resembles it, although differing in some particulars, may be given.

CASE XLIV.—*Fatty Degeneration of the Heart without Valvular Lesion.*

The Rev. Mr. P., aged thirty-five years, visited me on the 14th February, 1873. He was a curate, of very active habits and great zeal, and located in a mining district in the south of Ireland, where the vice of drunkenness prevailed to an alarming extent. This he undertook to combat with all his power. With this object in view, he established a temperance society, and hired a room, in which he lectured to the members on the evils of intemperance for several hours, three or four times in the week; pursued them into their haunts, and, being a strong and resolute man, frequently used physical force to drag them out of the public houses. On one occasion whilst so engaged he got very roughly handled. He suffered greatly in mind at the misfortunes of those whom he laboured to reform, and frequently felt quite exhausted after his exertions. About two years prior to his visit to me, he began to experience oppression of the chest with palpitation, and some difficulty of breathing, but without cough.

During the succeeding twelve months he had several attacks of syncope ; and three weeks before I saw him, he felt faint from the effects of a hard morning's work, and a rapid walk of some miles after breakfast. The heart, on this occasion, beat with great violence, and he was bewildered and wandered about the house without knowing why or where he went. This state lapsed into complete syncope in which it was thought he would have died, and during which, according to the report of his attendants, he was deadly pale. He declared, however, that neither in this attack, nor in any of those which preceded it, did he entirely lose consciousness.

At the date of his visit to me, the slightest exertion or emotion sufficed to bring on fluttering of the heart, oppression, and vertigo ; he had lost flesh, but was still corpulent, though flabby. Pulse 84, and regular but weak ; respiration regular and tranquil ; skin of an olive tint ; liver somewhat enlarged and tender ; bowels constipated ; urine depositing lithates at intervals ; no oedema ; no cardiac impulse to be felt ; first sound faint and dull, and heard best in normal situation of apex ; second sound morbidly clear at base, and in line of ascending aorta. No murmur anywhere to be heard, and no detectable rigidity or tortuosity of the superficial arteries.

The diagnosis made was as above stated, and the treatment consisted in ℞ij of liquor of strychnine, with grs. iiss of sulphate of quinine, thrice daily ; a pill containing grs. ij of extract of aloes, gr. j of extract of hyoscyamus, and gr. ss of extract of nux vomica every night, or every alternate night, as required. Moderate exercise on foot, short of fatigue, was enjoined, half a wineglassful of whiskey with water to be taken twice or thrice daily ; if threatened with syncope, a little brandy and water, together with a draught consisting of sulphuric ether ℞lx, and laudanum ℞xx, which he is to have ready at hand. Mental excitement, and the use of fatty food, was to be strictly avoided.

Two months later, Dr. O. Hanlon of Castlecomer, the Rev. Mr. P.'s ordinary medical attendant, reported to me that since his return to the country, in February, this gentleman continued to perform the duties of a very arduous mission, and had had no return of his previous symptoms.

In August, after a further interval of three months, I learned that he had been recently transferred to a new sphere of missionary labour.

CASE XLV.—*Fainting Fits; Œdema; Quick and Irregular Pulse; Hæmoptysis; Feeble Impulse and Systolic Murmur at Left Apex, at Lower Sternum, and in Ascending Aorta; Rough Systolic Murmur at Root of Pulmonary Artery; Death. Atheroma of Aorta; Mitral and Aortic Valves Thickened, and latter Calcareous; Heart Large and Covered with Fat, and Fatty Degeneration of its Substance (?) Right Lung Congested, and both Lungs partially Enveloped in False Membrane.*

Owen K., a travelling dealer in soft goods, aged sixty-eight years, admitted into Hospital, March 11th, 1865. Health reported good up to eleven weeks before, when he had an attack of bronchitis. Five weeks later, whilst getting out of bed to pass water, he felt a swimming in his head, and would have fallen had he not been supported. He remained in a state of half consciousness for forty-eight hours after this attack, and had another of a similar kind, but milder and shorter, consisting only of vertigo, on the Sunday preceding his admittance. Œdema of the lower limbs appeared seven weeks ago. He is pale; arcus senilis well marked; feet and legs much swollen, and feet thickly mottled with purpuric spots. Pulse 126, moderately full, and slightly irregular; respiration 24; cardiac impulse feeble. A loud bellows-murmur replaced the first sound at the left apex. A murmur of similar rhythm was likewise audible over the lower part of the sternum, and was traceable upwards in the course of the aorta to the second right chondro-sternal articulation, but less distinctly. The second sound was free from murmur, but ill pronounced, except above and to the right of the sternum, where it was distinctly heard. Respiration was feeble all over the chest, and associated with coarse crepitant râles over the bases of both lungs posteriorly. Cardiac murmur not audible behind. He declared that his breathing had never been irregular, nor was there, during the fainting fits, any cardiac distress or palpitation.



The diagnosis made at this date was regurgitation at the mitral, and obstruction at the aortic orifice, hypertrophy of the left ventricle, and a fatty state of the heart. To have nutritious diet and four ounces of wine daily, and a mild aperient as required.

13th. Pulse 102, slightly irregular, as is likewise the action of the heart. The second sound was accentuated behind the right second costal cartilage, where the systolic murmur was remarkably rough; slight *frémissement* over the heart during expiration; urine, sp. gr. 1.012, containing albumen in small quantity, and amounting to forty-eight ounces in twenty-four hours. To have tincture of the perchloride of iron and chloric ether, of each 3ij, with infusion of quassia to 3viij; a tablespoonful to be taken thrice daily. After a few days this medicine disagreed with the stomach, and compound iron mixture was substituted for it.

Still later (28th), citrate of iron and quinine was given in doses of grs. ij.

On the 30th, the pulse was 96, and regular; vertigo on assuming the erect posture. Over left apex a systolic murmur was heard, but no sound; over the base of the ensiform cartilage a systolic murmur and both sounds of the heart were audible.

On the 4th April he had an epileptiform fit, lasting half an hour, during which he lost consciousness and bit his lip; the pupils were dilated; the pulse was full, but not quick, and the cervical veins were engorged, but there was neither muscular spasm nor foaming at the mouth. Previous to the attack he was in a state of extreme nervousness, which he regarded as premonitory of it.

8th. Urine passed in large quantity, containing much albumen, and neutral in reaction, sp. gr. 1.012. Murmur transmitted into right carotid.

11th. Much improved; pulse 84, and strong; appetite and sleep much better; bowels regular, swelling of feet much diminished, and purpuric mottling has nearly disappeared.

13th. Had three attacks of hæmoptysis; viz., at 9, P.M., and at 1 and 6, A.M. In the second attack he threw up more than a pint of florid blood, and was nearly suffocated. Pulse 114, full; respiration 36; base of both lungs dull behind, with coarse crepitation audible, especially on the left side; feet not swollen,

but purpuric mottling now present in an extreme degree. He apprehends another attack in the course of to-night, which he says "will carry him off." To have grs. v of acetate of lead, and gr. iss of acetate of morphia, in water and distilled vinegar, every fourth hour. To be dry cupped over base of both lungs posteriorly, and to have cold drinks.

16th. Had a slight attack of hæmoptysis, accompanied with dyspnœa and excitement at a-quarter past 7, A.M.

25th. Very weak and unable to leave bed; no swelling or discoloration of feet; pulse 108, weak, but regular; respiration 36, strong and visible pulsation of carotids; a harsh murmur with the first sound, and very superficial, heard over the left third costal cartilage at its junction with the sternum, suspected to originate in, or upon the root of, the pulmonary artery. Other cardiac phenomena as before.

28th. Is incoherent and sinking. Over the sternal end of the left third costal cartilage, but loudest half an inch below and to the right of this point, a loud grating murmur is heard with the first sound.

He died on the 29th April, at 1, P.M.

The lungs, heart, and great vessels were removed a few hours after death, and examined on the 2nd May. There was much fat in the anterior mediastinum and around the pericardium; there was likewise a large deposit of fat on the exterior of the heart. Three "white spots" of considerable size were discovered; one about the size of a crown-piece on the anterior surface of the right ventricle, another somewhat smaller on its inferior surface, and a third as large as a florin-piece on the anterior surface of the pulmonary artery, at its origin from the right ventricle. This, no doubt, was the cause of the rough grating murmur heard on the 25th and 28th April, over the sternal extremity of the left third costal cartilage; right cavities not altered; pulmonary artery and its valves healthy; left ventricle greatly hypertrophied, and its cavity diminished; mitral valves much thickened, but no deposit upon them. Aortic valves competent to retain water poured into the aorta, but rough with calcareous deposit. Lining membrane of aorta bright red, and interspersed with yellow patches of atheroma. It was rough, and in a more advanced state of

atheromatous change, at the origin of the great vessels, and in the lower wall of the transverse portion of the arch. The apices of both lungs were encased in a thick layer of false membrane; that on the right side was several lines thick, and the parenchyma of the lung beneath was solidified, apparently by contraction of the enveloping false membrane. The lower portion of the right lung was congested, and likewise covered by a layer of false membrane. Other organs not examined. Owing to an oversight, the muscular substance of the heart was not examined microscopically.

CASE XLVI.—*Dyspnœa ; Œdema ; Quick and Irregular Action of the Heart ; Venous Pulsation in the Neck ; and, eight days before Death, Left Hemiplegia and Partial Aphasia ; Displacement of the Apex of the Heart to the Left, and the First Sound Abnormally Clear ; no Murmur. Extravasation of Blood into the Right Anterior Lobe of the Cerebrum, and Plugging of the Right Middle Cerebral Artery ; Granular Degeneration of the Kidneys ; Enlargement and Fatty Degeneration of the Heart ; Dilatation and Thinning of the Right Ventricle ; Valves all Sound.*

Michael T., aged sixty-six years, a porter, of temperate habits, was admitted into hospital, September 25th, 1865. About two years previously, having been much exposed to wet and cold, he complained of cough. Twelve months later, his breathing became very much oppressed, and his feet began to swell.

On examination, his condition was found to be as follows: There was œdema of the face and lower extremities; the action of the heart was quick (120 in the minute), and irregular; the apex was displaced to the left, and the first sound was abnormally clear. There was dulness on percussion, and inspiratory wheezing, over both lungs posteriorly. Marked pulsation of the superficial cervical veins, synchronous with ventricular systole, was visible during inspiration. Urine diminished in quantity, acid in reaction, sp. gr. 1.010, and without a trace of albumen. To have thrice daily a tablespoonful of a mixture consisting of Iodide of potassium, 3iiss; Acetate of potash, 3ij; Infusion of gentian, to 3viij.

October 3rd. Breathing less oppressed; urine increased in quantity (a quart last night) and neutral in reaction. On the following night the urine passed amounted to two quarts, and the night of the 5th the quantity was still greater. There was less tumefaction of the face, and the œdema of the legs had quite disappeared.

22nd. Whilst returning from the water-closet he became suddenly hemiplegic on the left side of the body; the left side of the face was but partially affected, the patient being able to close the eye and corrugate the forehead on that side. Consciousness was perfect, as was likewise the power of deglutition, but there was almost total inability to articulate. No paralysis of the bladder or rectum.

The patient died on the 30th October, and the body was removed for dissection to the Medical School, Cecilia-street, where a careful examination of it was made. The right hemisphere of the cerebrum was soft, and in excess of the left hemisphere in volume. In the upper part of the right anterior lobe there was a large blood-clot, from which blood had been diffused into the surrounding brain substance, breaking up the latter within a radius of an inch and a-half. The corpus striatum and other parts were unaffected. The right middle cerebral artery was so plugged with fibrin, that the ordinary coloured injection used in the dissecting room could not pass, under high pressure. The kidneys were smaller than natural, and the cortex of the left was so attenuated that the Malpighian pyramids had all but come to the surface. The heart was large, soft, and so flabby, that when placed upon the table, it expanded by its own weight, and became quite flat; it was likewise torn with great facility. There was only a very slight deposit of superficial fat. The right ventricle was dilated, and its walls were remarkably thin. The left cavities and all the valves were normal. Under the microscope, the fibres of the left ventricle exhibited lines of oil dots in place of the normal fibrils. In one situation only the transverse striæ were visible.



CASE XLVII.—*Recurrent Angina and Hæmoptysis; Irregular and Intermittent Pulse; Œdema; Albuminuria and Slight Jaundice; Feeble Cardiac Impulse and Sounds; Irregularity of Breathing; Gangrene of the Foot and Leg on one Side; Coma; Death. Diagnosis: Dilatation and Atheromatous Change of the Aorta; Enlargement and Fatty Degeneration of the Heart, and Accumulation of Fat upon its Surface; Atheromatous or Calcareous Change of the Smaller Arteries, and Thrombosis of those of the Right Foot and Leg.*

J. N. W., a medical man in extensive practice, aged sixty-one years, large and corpulent, consulted me October 22nd, 1866. Five years previously, whilst ascending a steep incline, he complained of shortness of breath, and spat a little blood. Two years later he coughed up a considerable quantity of blood, and was bled from the arm to eight ounces. Three weeks previous to my visit, after rapidly ascending a flight of stairs to attend an urgent call, he suffered extreme dyspnoea, was obliged to sit down and have the window thrown open in order to catch his breath, and spat some blood.

He had occasionally suffered from derangement of liver and kidneys, the urine being loaded with lithates; and once, very recently, from acute pain in the right lumbar region, which lasted but a short time, and terminated with a copious flow of urine loaded with lithates.

For some days prior to my visit he had been suffering from paroxysms of dyspnoea, accompanied by a feeling of great oppression in the region of the heart. The face and conjunctivæ were slightly jaundiced; liver much enlarged and tender; and pressure at the epigastrium aggravated the feeling of oppression in the region of the heart. The stomach and bowels were distended with flatus; the bowels were confined, and were moved only by means of aperients. The stools were ochrey in colour; urine, passed in small quantity, was clear and amber-coloured at my first visit, but on the following day, fawn-coloured and muddy; in sp. gr. it was 1·020, acid, containing lithates, and a small proportion of albumen. Pulse 114, small, and rather

weak, but regular ; according to his own report it has been occasionally intermittent. Heart's action obscure ; no impulse perceptible, and precordial dulness extended generally. The first sound was ill pronounced and masked, but free from murmur ; second sound in some degree disintegrated and muffled ; no impulse anywhere discoverable over the chest. The radial pulses and the pupils were respectively equal, and no dysphagia existed. In the course of the preceding few nights he had got some hours of broken sleep in an easy chair, with the help of 3ss of liquor of the hydrochlorate of morphia. To take daily six pills, containing each gr. iss of squill and of mercury with chalk, and grs. ij of dried soda. A dozen leeches to be applied, in two relays, over the liver, and then a warm poultice. Mercurial ointment was subsequently applied freely in the region of the liver. The use of liquids was restricted. Beef tea and bread crumb were ordered as food, and a small quantity of whiskey and water was given occasionally, when debility seemed to demand it.

25th. 4 P.M., pulse feeble, and occasionally intermitting. In the course of this evening, whilst in bed and at rest, he had a slight attack of vertigo, accompanied with momentary loss of consciousness. Sir D. Corrigan saw him, and coincided in my view of the case.

On the 27th there was slight mercurial fetor ; he had several hours of undisturbed sleep the preceding night, after a large blister had been applied over the sternum. To have grs. ij of calomel every sixth hour.

29th. Considerable œdema of lower limbs ; had a severe paroxysm of dyspnœa in the course of the preceding night.

November 1st. Since last report he has been improving steadily. He slept well the preceding night in the horizontal posture ; abdomen much reduced in girth, and hepatic tenderness all but removed. Pulse regular, but weak ; tongue clean ; urine passed in the preceding twenty-four hours amounted to forty ounces ; it was clearer ; appetite and spirits improved ; mercury stopped, and patient allowed chicken to-day.

It had been arranged, at the patient's urgent request, that he was to leave town on the 3rd of November for his residence in a midland county ; but on the preceding night he became alarmingly

ill, having been seized with a paroxysm of dyspnœa of unusual severity.

On the 3rd, the pulse was weak and intermittent, and the œdema of the lower extremities was increased.

On the 4th, he became livid, pulseless, and slightly incoherent, and was again visited by Sir D. Corrigan in consultation with myself and his son, a medical officer in the army.

On the 5th, large bullæ had formed on the feet and legs ; there were repeated paroxysms of dyspnœa, in which the breathing became gradually more and more rapid, and then as gradually subsided to the normal standard ; pupils greatly contracted.

7th. Pulse irregular, intermittent, and weak ; pupils contracted ; heart-sounds obscure and feeble, but without murmur. He is now unable to get into bed, obtaining only a few snatches of sleep in an arm chair. Feet, legs, and thighs highly œdematous, depressed in temperature, and, as high as the knees, studded with large bullæ containing clear serum. Cannot take food of any kind, except a little egg-flip ; is much troubled with hiccough and bilious vomiting.

During the 6th and 7th, there was copious diuresis without deposit, the quantity passed in twenty-four hours amounting to eighty ounces.

Diuresis continued on the 8th ; the bullæ had given way, and there was a free discharge of serum from them. The pulse was irregular, intermittent, and barely to be felt, yet the patient declared he felt better.

On the 9th, he was comatose and pulseless ; the thighs were greatly swollen, and on the inner aspect of the right, there was a large patch of inflamed surface, of a yellow-red tint.

On the 11th, the right foot and leg, half way to the knee, were in a state of gangrene ; but no further change had taken place in the thigh ; he was conscious ; had within the preceding twenty-four hours repeated attacks of gasping dyspnœa, attended with laryngeal stridor and projection of the eye-balls. I witnessed three such seizures within a period of ten minutes. They came on without any assignable cause, whilst the patient lay in bed, but lasted only a few seconds.

In the afternoon of this day he died, and no examination of

the body was made. The diagnosis given in the title of this case, though unsupported by the evidence of *post mortem* examination, is, I think, fully warranted by the symptoms and signs exhibited, as well as by its analogy to other cases published in this list, in which the diagnosis was confirmed by the crucial test of dissection.

CASE XLVIII.—*Derangement of Stomach, and Anomalous Nervous Symptoms; Sudden Death. Congestion of Lungs; Enlargement and Fatty Growth upon, and Advanced Fatty Degeneration of, the Heart; Excentric Hypertrophy of the Left Ventricle; Dilatation and Advanced Atheromatous Change of the Aorta.*

Robert C., aged forty years, a respectable man, but reduced in circumstances, who had been for some weeks attending at the Mater Misericordiæ Hospital as an extern patient for derangement of bowels and anomalous nervous symptoms, presented himself at the usual hour on Wednesday, July 14th, 1869, in his ordinary state of health, and making no complaint of any particular ailment. On the evening of that day he was carried to the hospital by a person who had found him in an adjoining field, in a state of insensibility. When received into hospital he was quite unconscious, pallid, cold, and pulseless; he died shortly after his arrival.

The lungs were congested, but resonant. The heart large, and covered, on right side especially, with a thick layer of fat; it weighed eighteen and a-half ounces, and expanded by its own weight when laid upon the table. The right ventricle was dilated, but not thinned, and the superficial fat had penetrated nearly to the internal surface at the apex. The left ventricle was dilated and hypertrophied; several of the fleshy columns were dotted over with large yellow spots, and a section of one of them, which presented this appearance over about one-eighth of an inch of its length, exhibited its entire substance, all but a mere shell, converted into soft fat. Microscopically examined, the walls of the heart were found in an advanced stage of fatty degeneration.\* The valves were all healthy and competent, with

\* By an oversight, the particulars of the microscopic examination were not noted.



the exception of the anterior-right curtain of the mitral, which was a little thickened on its free margin. The arch of the aorta, especially the ascending portion, was much dilated, rugose on its inner surface, and mottled with atheroma; on section of the vessel, this change was seen to engage the middle coat in large patches. The lining membrane had given way at one or two points near the valves, and had become detached to a slight extent from the middle coat.

CASE XLIX.—*Œdema of the Lower Limbs, followed by Gangrene; Enlargement of the Liver; Weak Impulse; Rapid and Irregular Action of the Heart; Hæmoptysis; Intermittent Musical Systolic Murmur at the Apex of the Heart; Venous and Arterial Throbbing in the Neck; Sudden Death. Enlargement of, and Fatty Accumulation upon, the Heart, and Fatty Degeneration of its Substance; Valves all virtually Sound; Cirrhosis of the Liver; Pulmonary Apoplexy.*

Richard B., aged sixty-nine years, a cabinet-maker, reputedly temperate, was admitted into the Mater Misericordiæ Hospital on the 16th of January, 1868. He was then generally anasarcaous; the lower extremities were greatly distended, and the fingers and toes were livid. The liver was uneven on the surface, and tender to pressure. The radial pulse was irregular, and so small and feeble that it was not calculable. The action of the heart was remarkably tumultuous and irregular, and varied in rate from 156 to 180 in the minute. There was strong venous and arterial pulsation in the neck. It was impossible to determine accurately the extent of precordial dulness. At the apex, and faintly at the base, but not transmitted in the course of the aorta, a remarkable musical murmur was heard with the first sound of the heart, but only with every fourth or fifth pulsation. The second sound was normal, as regarded quality, in both situations, but remarkably weak. The respiration was quick, 36 in the minute, and attended with dry râles all over the chest. Urine, sp. gr. 1·022, acid, and free from albumen, but loaded with lithates.

On the 18th he began to expectorate blood, which was occa-

sionally mixed with mucus. The pulse now became imperceptible at the wrist; the apex-murmur ceased to be audible; it was, however, again heard on the 21st, 22nd, and 23rd, after two days' silence. It was now much fainter than before, and of a squeaking character, not very dissimilar to the noise elicited by blowing through a reed.

On the 25th, a large bulla, the size of a crown-piece, appeared on the dorsum of the left foot. The feet were quite cold, yet the man complained of a burning sensation in them, and, in order to alleviate that sensation, he insisted on allowing them to hang out of bed.

On the evening of the 25th, having a call to the night-chair, he got out of bed, and a few minutes afterwards he was found dead in the sitting posture. He had been for some time previously incoherent.

A *post mortem* examination revealed the following condition of organs. The sinuses of the dura mater and the cerebral veins were remarkably injected. The lungs, with the exception of the base of the right, in which there was a mass of apoplectic extravasation, were healthy. The heart was enlarged, weighing eighteen ounces, with a considerable deposit of fat on its surface. The right chambers were dilated, and in a special degree the right ventricle; the tricuspid orifice was greatly enlarged. The left ventricle was scarcely dilated, but its walls were somewhat thickened. The valves, both mitral and aortic, were virtually normal, and apparently competent. At the root of the aortic valve some spicula could be felt by the finger, but they were manifestly incapable of impairing its efficiency, or of obstructing the current of exit; the mitral valve was of a deep yellow tint; the liver was enlarged, and in the early stage of cirrhosis; the kidneys were likewise enlarged, the cortex was thick and yellow, and the tubes contained oil dots in large numbers. The walls of both ventricles were in a state of fatty degeneration; those of the right in a more advanced stage than those of the left. There was a good deal of fatty deposit between the fibres.

The existence of mitral systolic murmur in this case, without valvular lesion, is, in my opinion, to be explained by the state of tissue-degeneration, and consequent yielding of the

walls of the left ventricle, as in some of those previously given. Its musical character was probably due to a flake of fibrin appended to the edge of the valve, and subsequently detached and broken up. But of this there is no positive evidence.

Of the interesting features presented by the preceding case, the following is still further illustrative.

CASE L.—*Dyspnœa and Irregular Breathing ; Palpitation ; General Dropsy ; Hæmoptysis ; Enlargement of the Liver, and Slight Jaundice ; Failure of the Pulse ; Rapid and Irregular Action of the Heart ; Apex-Systolic Murmur ; Basic Double Murmur ; Gangrene of the Right Arm ; Death. Enlargement of, and Fatty Deposit upon, the Heart, and Fatty Degeneration of its Substance ; Dilatation and Hypertrophy of the Left Ventricle ; Dilatation, Atheromatous Change, and Inflammation of the Aorta ; Arteritis extending into the Carotids, Right Subclavian, and Axillary Arteries ; no Disease (virtually) of the Valves, and no Plugging of the Arteries ; Fatty State of Liver ; Congestion of Right Lung.*

John W., aged sixty years, a gunsmith by trade, was admitted into the Mater Misericordiæ Hospital on the 9th October, 1867. He had been temperate, and had not had rheumatism. Up to six months previously, he had enjoyed uninterrupted good health; and then, for the first time, his respiration became quick on ascending a flight of stairs, and there was palpitation. A month later, swelling of the feet and legs appeared.

At the date of admittance, there was very considerable swelling of the feet, legs, and thighs; a rather remarkable superficial venous engorgement of the dorsal surfaces of the feet and lower part of the legs; and a slight jaundiced tint of the face and conjunctivæ. There was considerable effusion into the peritoneum; the liver was enlarged considerably: it extended nearly to the umbilicus. The pulse was 96, irregular, and intermittent. He occasionally sighed, and respiration was accelerated at intervals, even as he lay quietly in bed. The precordium was decidedly dull from the right margin of the sternum to an inch and a-half external to the nipple-line. In this latter situation, a distinct

bellows-murmur was heard replacing the first sound of the heart. It was audible as far as the left axilla, and likewise at the inferior angle of the left scapula. At the apex, the second sound was obscure, but still audible. Over midsternum, a systolic murmur, but less pronounced than that at the apex, was likewise audible, and here, also, a diastolic murmur was heard. There was visible pulsation of the radial and carotid, but not of the temporal arteries. Urine passed in moderate quantity, sp. gr. 1.015, acid, and with only a trace of albumen.

Up to the 19th, he continued to improve under a treatment consisting of tonics and diuretics; but towards the end of October, the penis and scrotum became swollen, an exceedingly unfavourable symptom in such a case. The penis was punctured, and on the following morning the discharge of serum had been so great, that it had filtered through the bed, on to the floor. Great relief was derived from this procedure, which was subsequently repeated. He soon began to sink; had repeated paroxysms of dyspnoea; blood was mixed with the sputa, and the lower part of the left side became dull on percussion.

On the 19th, a blush was observed on the left elbow.

On the 20th, this had extended to the shoulder and to the wrist, was attended with great tumefaction, and large vesicles had formed on the surface, which presented a lemon-yellow tint.

On the 21st, the tumefaction and redness, now of a dark hue, had extended to the right side of the chest and neck.

On the 22nd, the entire of the right upper extremity was tense, and large bullæ had formed on the elbow. The radial pulse was slow, only 72, whilst the heart beat at the rate of 156 in the minute. Cutaneous sensibility was impaired, as no pain was experienced on pressure.

He died on the morning of the 23rd.

The right upper extremity was in a state of incipient gangrene; the cuticle had been detached from the greater part of the surface. All the great serous cavities were full of liquid. The right lung was consolidated in its lower half, was dark and dry in section, and this part of the organ sank in water; the remainder of the right lung was œdematous. In the apex of the left lung,



there was a mass of calcified tubercle, and the surface of the lung corresponding to this was puckered. The heart was greatly enlarged, weighing, with one inch of the aorta and pulmonary artery attached, thirty-two and a-quarter ounces. It was fatty on the surface, and presented two "milk spots;" viz., one, remarkably white, on the anterior surface of the right ventricle about the centre; and another on the anterior surface of the left ventricle near the apex. The right chambers contained much dark coagulated blood. The tricuspid orifice was greatly dilated. The right ventricle was dilated and thickened in some degree, and its sinus, at the origin of the pulmonary artery, was dilated into a large pouch, presenting the appearance of a distinct chamber. Pulmonary artery dilated, but valves competent. The left chambers contained some dark clotted blood. The mitral valve was thickened, but not otherwise altered; it was competent to close the orifice, as proved by the water-test. The left ventricle was greatly thickened and dilated, and the left auricle dilated only. The walls of the left ventricle were nine-sixteenths of an inch thick at the apex, three-fourths of an inch at the middle, and two-fifths of an inch at the base. The cavity measured four and three-quarter inches from the root of the left segment of the mitral valve to the apex, and from the septum to the left wall, in the state of collapse, two and a-half inches.

Microscopically examined, with a power of 222 diameters, the muscular fibres of the left ventricle were found converted into strings of oil dots, which, after treatment with strong acetic acid, were seen arranged in unbroken linear series. No trace of transverse striation was anywhere visible. The aorta was dilated, atheromatous in patches, and of a deep scarlet tint throughout the arch. The aortic valves were healthy, but, owing to the dilated state of the vessel, they were inadequate. Water poured into the aorta flowed slowly into the ventricle through a small aperture in the axis of the opening. The deep scarlet tint of the aorta extended into both carotids, the right subclavian, and the axillary arteries; it was nowhere removable by washing. No embolus was found in any of the arteries of the right upper extremity, all of which were carefully examined.

The liver was enlarged, and contained much fat in the form of oil globules, both in the hepatic cells and interstices; it sank in water. The gall bladder was empty. The spleen was of normal size, and apparently healthy.

In relation to this case, the following remarks were made on the occasion of submitting the details, together with the diseased organs, to the notice of the Pathological Society of Dublin.\* “The most interesting features in this case were, first, the existence of mitral regurgitant murmur without mitral valve-disease adequate to its production, as proved by dissection.

“The explanation of such an occurrence, which, in the absence of valvular lesion or inadequacy, must be attributed to yielding of the walls of the left ventricle during systole, was to be found in the state of tissue-degeneration of that chamber.

“Secondly. The existence of two murmurs at the base, without structural lesion of the aortic valve; namely, a systolic murmur produced by a rough state of the interior of the aorta immediately above the valves, where atheromatous change was most advanced; and a murmur of diastolic rhythm, produced by axial reflux, from incompetency of the valves to occlude the dilated orifice.

“Thirdly. The existence of aortitis, and arterial inflammation extending into the carotids and main artery of the right upper extremity, followed by gangrene of the right arm, of which it afforded the only rational explanation in the absence of arterial plugging.

“Fourthly. The existence of hypertrophy of the heart, to the extent of nearly threefold development, without valvular disease, and dependent upon disease of the aorta.

“Consecutive to hypertrophy there was fatty degeneration and softening of the ventricles, and mitral regurgitation by excentric yielding of the left ventricular walls, engorgement of the lungs, and general venous congestion and dropsy.

“Fifthly. Cardiac asthma or panting, and irregular respiration at intervals, associated with an atheromatous and dilated state of the aorta, of which I believe it to be pathognomonic.

\* December 7th, 1867, *Proceedings*, vol. iii., part ii., new series, p. 233.

"Lastly, the existence of jaundice. In dropsy of cardiac origin, and connected with hepatic engorgement as its ostensible cause, I am disposed to regard jaundice as evidence of fatty disease of the liver, and inferentially, of the heart also."

CASE LI.—*Paroxysmal Dyspnœa; "Ascending and Descending" Respiration; Palpitation; Rapid Action of the Heart, and Failure of the Radial Pulse; Œdema and Paralysis of the Left Arm; Cardiac Sounds Clear over Lower Sternum; Systolic Murmur; Death by Asthenia. Heart Enlarged, and Fatty on the Surface; Right Chambers Normal, and Structurally Sound; Left Ventricle Thickened, but not Dilated, except at Apex, where it was greatly Thinned; Fatty Degeneration of its Substance; Mitral and Aortic Valves Thickened and Inadequate; Aorta greatly Dilated, and slightly Atheromatous.*

James K., aged sixty-two years, a carpenter, temperate, admitted August 7th, 1869. For the last year he has suffered from slight palpitation and occasional shortness of breath, and eight weeks prior to admittance a beam of wood fell upon him, and gave him a great shock; since that date he has been more incommoded by palpitation and dyspnœa. A week since, whilst proceeding to his work one morning, he was suddenly seized with dyspnœa, felt as if he were dying, and was forced to return home. His breathing has been since constantly embarrassed, with occasional paroxysmal aggravations, which come on in the morning whilst he lies in bed; he then feels as if his breathing were about to cease. For the last few days he has spent much of his time at the open window, in order to obtain a free supply of air.

The face is slightly jaundiced. There is great dyspnœa. No œdema. Urine passed in small quantity, and frequently. Pulse arhythmically irregular, about 96 in the minute, but difficult to register owing to the existence of many abortive and scarcely perceptible pulsations. Heart's action foetal in character, arhythmically irregular, and varying from 168 to 192 in the minute; cardiac sounds morbidly clear and ringing over lower sternum, with a soft murmur accompanying the first sound. To have

Tinct. of digitalis, ℥<sub>x</sub>; Tinct. of perchloride of iron, ℥<sub>xx</sub>; Spirit of nitrous ether, ℥<sub>xl</sub>, every third hour; and at night, Hoffman's liquor, ℥<sub>xxx</sub>, with liquor of hydrochlor. of morphia, ℥<sub>xv</sub>, in camphor water.

14th. Has diarrhoea, with sickness of stomach. Medicine to be stopped, and grs. x of aromatic chalk powder to be given.

16th. Is much improved; can sleep in the horizontal posture; no diarrhoea. To have quinine and sulphuric ether, and to be allowed to get up.

25th. Confined to bed; radial pulse, 96, rate of 192; respiration "ascending and descending," *i.e.*, in the recumbent posture it became gradually quick and gasping, till it attained a rate of cardiac action 63 in the minute; this, which was its greatest rate, was reached in the course of about two minutes. It then as gradually subsided till it ceased altogether, remaining suspended for *twenty-two seconds*. At this rate, there would be little more than three respirations in the minute.

27th. No cardiac murmur to be heard. Pulse nearly suppressed in left arm, which is paralysed and swollen. Breathing, as last noted. It was particularly observed that during the period of apnoea there was no alteration of the heart's action, or of the radial pulse, in regard to rate or otherwise. He is only partially conscious, and is manifestly sinking.

Died quietly on the 30th.

The heart was enlarged, somewhat globular in figure, fatty on the surface, and blunted at the apex, which was formed entirely of the left ventricle. Attached to the apex there was a thick and tough shred of false membrane. The weight of the heart, with two inches of the aorta and one inch of the pulmonary artery attached, was twenty ounces. The aorta was enormously dilated, admitting readily the first three fingers introduced edgewise; it was slightly atheromatous. Two of the curtains of the aortic valve were thickened and nodulated on the free edge and lower surface, so that a slight dehiscence existed between them when approximated, permitting very slow passage of water from the aorta into the ventricle. The orifice of the aorta was not dilated. The right chambers were normal, and contained some decolorized clot, disposed in globular masses. The left auricle was much di-



lated. The mitral valves were thickened and agglutinated at the edges, so as to present a permanent opening of the figure of a button-hole, which, however, admitted the points of two fingers. The valves were incompetent. The left ventricle was thickened, but not dilated, except at the extreme apex, where it was so thin over a space equal to the point of the finger, that the thumb placed externally at a corresponding point, was readily felt through the ventricular wall. The muscular fibres of the left ventricle, examined microscopically, were found in an advanced state of fatty degeneration; but, by an oversight, I did not preserve a note of their precise appearance. There was some serous effusion in the right pleura. The abdomen was not examined. The structural integrity and normal condition generally of the right chambers of the heart, afforded an explanation of the clear and clicking character of the cardiac sounds over the lower sternum, and the absence of dropsy. The irregularity of action was a reflex of that which existed on the left side. The "ascending and descending" character of respiration, followed by a period of apnoea, was here exemplified in the highest degree. The aorta was greatly dilated. The close connexion between the respiratory phenomenon just mentioned, on the one hand, and the dilatation of the aorta and weak state of the left ventricle upon the other, I have already dwelt upon in the preceding pages.

Similar to the last case, in many respects, is the following.

CASE LII.—*Dyspnœa; Dizziness; Respiration of "Ascending and Descending" Rhythm; Rapid and Irregular Action of the Heart; no Œdema; Double Murmur at the Base of the Heart and in the Arch of the Aorta, the Systolic being transmitted into the Carotids; Diastolic Murmur, and Quasi-Presystolic Murmur at the Apex; Drowsiness; Death. Lungs Emphysematous and Congested; Heart Enlarged and in a state of Fatty Degeneration; Mitral Orifice slightly Narrowed, and Beaded with Vegetations of Lymph; Aortic Valves Thickened, and Incompetent; Aorta Dilated and Calcareous.*

James D., aged sixty years, a labourer, of temperate habits,

was admitted October 5th, 1869. His health was good up to three years previously ; he then began to suffer from "gnawing" at the pit of the stomach, and weakness. Since then he has had dizziness in the head three times, obliging him to support himself against some fixed object, and giving him the sensation and appearance of being under the influence of alcohol. Three weeks ago his breathing began to be short. He never spat blood, and never suffered from rheumatism.

*State on admission.* There was no cedema. Pulse arhythmically irregular, and varying from 72 to 84. There were frequently two beats of the heart in rapid succession, both being represented in the radial pulse.

The carotid, tibial, brachial, and radial arteries pulsated visibly ; the two latter being remarkably tortuous. The apex-pulsation of the heart was behind the sixth rib, half an inch outside the nipple-line ; here, the first sound was prolonged, and associated with a murmur of quasi-presystolic rhythm. In this situation, likewise, a loud bellows-murmur took the place of the second sound, and was distinctly audible in the left axilla. The second sound was not intensified in the pulmonary artery.

At the base, a loud double bellows-murmur was audible, the diastolic being the louder ; both were transmitted into the arch of the aorta, and the systolic likewise into the carotids. Both murmurs were transmitted some distance towards the apex, but were nearly lost at the seat of the apex-beat. To have Hoffman's anodyne, 3ss ; and liquor of hydrochlorate of morphia, ℥x, at night ; and tincture of perchloride of iron, and chloric ether, of each ℥x thrice daily.

For a fortnight the patient seemed to improve ; the pulse was more steady, he slept better, took food in sufficient quantity, and the kidneys acted well. The medicine, having disagreed with the stomach, was then suspended, and replaced by quinine and sulphuric ether.

30th. Paroxysmal breathing, alternating with intervals of apnoea in the recumbent posture, were to-day for the first time observed ; the transition from the one to the other being abrupt. Pulse very weak and intermitting ; pupils dilated ; patient somnolent and incoherent. The diastolic murmur at the apex was

very loud and blowing, replacing the second sound, and extending nearly to the end of the long pause. Its terminal portion was rough in quality, resembling in some degree the murmur of mitral contraction, but not continued *up to* the first sound. Double murmur faintly audible at the base, but very distinct throughout the arch of the aorta. Was moribund.

31st. He was unconscious. There were alternating periods of apnoea, and of paroxysmal and suspirious breathing, the former occupying twenty-four, and the latter twenty-six seconds. During these alternating changes of respiration, the pulse and the action of the heart underwent no change, and the transition from the state of apnoea to that of paroxysmal dyspnoea, was less abrupt than previously, being accomplished within a period of about one second.

He died at 8, P.M., and the body was examined on the following day.

The lungs were emphysematous and congested posteriorly. The heart was enlarged, weighing, with one and a-half inch of the aorta, and an inch of the pulmonary artery attached, nineteen ounces. The right ventricle was very thin, its internal and external layers being yellow, whilst the intermediate portion was of the natural hue. Under the microscope, the internal layers showed fibres in the stage of granular degeneration, and others in that of obsolescence. One of the latter measured only the 2,664th part of an inch in diameter.

From the upper part of the right ventricle a remarkable conical appendage of fat projected; it lay over the root of the aorta, and was about two inches long, and one inch broad at its base. The left ventricle was thickened; its walls measured half an inch at the base, and under the microscope its fibres exhibited some oil dots; but the transverse striæ were distinct.

The right auriculo-ventricular orifice was dilated, and both it and the right chambers were partially occupied by a large decolorized mass of fibrin. The tricuspid and pulmonary valves were healthy. Both auricles were normal; the mitral orifice was somewhat less than the ordinary size, admitting only the tips of two fingers. The mitral valves were competent, and in all respects normal, save that at the attached margin, and on the

auricular aspect, there was a circlet of warty vegetations of about the size of duck-shot; they were soft, partially transparent, and smooth on the surface. The aortic valves were slightly thickened, and shrivelled at the free edges; they admitted of axial reflux. In one of the sinuses of Valsalva, and partially attached to the wall of the vessel, there was a rugged mass of calcareous matter about the size of a pea. The orifice of the aorta was of normal size, but the arch was much dilated, admitting the tips of three fingers; on its internal surface there were visible a few yellow dots of incipient calcareous formation.

The only feature in this case which demands special notice, is the existence of diastolic apex-murmur not due to aneurism of the heart. It is, indeed, the only genuine example of the kind which has come under my notice.

I have, in several instances, met with a murmur of postdiastolic rhythm, *i.e.*, a murmur *appended* to the second sound; this is peculiar to extreme mitral stenosis. But a murmur *replacing* the second sound, and having its source at the apex, is so rare, that the dissection of a body in which its existence was verified clinically, possesses more than ordinary interest.

In considering the cause of this phenomenon, the basic murmurs must be excluded, because it is distinctly stated that they both fell short of the apex-point; whereas it was here and in the left axilla only, that the murmur in question was to be heard. The mitral orifice was in some degree contracted, both relatively to the left chambers, and absolutely; and on the auricular aspect the valve was beaded with a string of wart-like vegetations. To this twofold condition, conjoined with suppression of the second sound in the aorta, the substitutive murmur of aortic inadequacy not reaching the apex, I am disposed to attribute the mitral diastolic murmur.

In the great majority of examples of mitral stenosis, a second sound is heard at the apex; it there serves, though in a subordinate degree, to gauge the rhythm of the presystolic murmur, which usually succeeds it at a long interval. Occasionally, however, this murmur is broken up into two fragments; one appended to the second sound, and constituting a true postdiastolic murmur; and the other prefixed to the first sound, and repre-



senting the presystolic murmur which is alone heard in ordinary cases. Between these, there is an interval of silence. The fragments vary as to relative length in different cases; but usually the postdiastolic element exceeds the presystolic in this respect. It may even happen that the presystolic element is all but suppressed, it being distinctly audible only during periods of excitement of the heart; and at other times requiring strained attention in order to detect it (see cases of the girls Mary E. C., Mary F., and Thomas D., in "mitral stenosis"). If, in such a case, no second sound is audible at the apex, the diastolic gauge being thus wanting, the more prominent murmur, or, perhaps, the only one recognized, may be readily mistaken for a murmur of true diastolic rhythm. This I believe to have happened in the case under discussion. According to the view now stated, the murmur was in reality not diastolic, but postdiastolic in rhythm; having been misinterpreted, owing to the absence of the ordinary chronometric standard, namely, a second sound.

CASE LIII.—*Delirium Tremens; Quick and Feeble, but Regular Pulse; no Cardiac Impulse to be felt; Sounds Obscure, but unattended with Murmur; Sudden Death by Syncope. Congestion of the Lungs; Enlargement of the Heart; Dilatation of the Left Ventricle; Fatty Growth upon, and Granular Degeneration of, the Substance of the Heart.*

— F., aged thirty-four years, a large, fat, and flabby man of intemperate habits, was admitted into hospital, February 1st, 1870, in *delirium tremens*. For some weeks preceding January 27th, he had left off the use of stimulants; but on that day he drank freely, and shortly afterwards exhibited symptoms of *delirium tremens*.

When admitted he was tremulous and restless, and had not slept for three nights previously. The conjunctivæ were injected, and the pupils contracted, the tongue slightly coated, and the bowels confined. Pulse 110, soft and weak, but regular; skin cool.

On the following day it was found that he had not slept; he

had raved incessantly through the night, and several times got out of bed. Pulse remarkably weak and failing; no cardiac impulse to be felt, and sounds obscure, but free from murmur. To have gr. ss of morphia in two ounces of sherry every third hour, and plenty of liquid nutriment.

After the third draught he slept four hours, and on the morning of the third, whilst taking some beef-tea after awaking, he suddenly died of syncope.

The lungs were large in volume, and generally congested. The heart was covered with a thick layer of fat, and so soft, that when laid on the table it expanded by its own weight. It weighed nineteen and a-half ounces, and contained dark fluid blood, with one imperfect coagulum. The valves were all healthy. The left ventricle was dilated, but not thickened.

Examined microscopically, the muscular fibres exhibited transverse striation, with large blebs of fat floating over the field. A portion of the internal surface of the left ventricle, which was of a lighter colour than the remainder of the wall, exhibited fatty degeneration of the fibres in the granular stage with an absence of striation.

CASE LIV.—*General Debility; Attacks of Syncope; Visible Pulsation of Arteries; Double Murmur at Base of Heart, and Single Systolic Murmur in the Carotids; Diagnosis; Hypertrophy and Fatty Disease of Heart; Disease of the Aortic Valves; Dilatation and Roughening of the Aorta.*

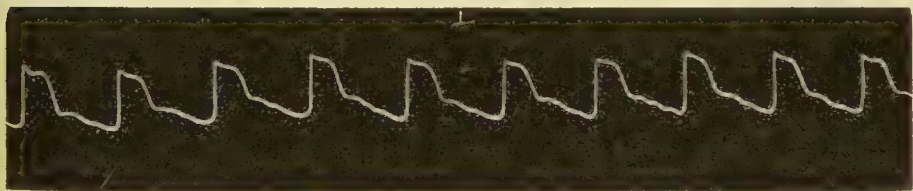
James D., a stout-built man, aged forty-nine years, a coachman by occupation, was admitted into hospital, June 27th, 1867. He had been intemperate, but never had rheumatism, or any other serious illness. About three weeks previously he began to lose power in all his limbs, but without experiencing any unusual sensations in them. There was wasting of the muscles, but no want of control over them. At the same time, he began to experience "a lightness" in his head, and his sight was obscured at intervals. A week previous to admission, whilst walking in the street, he felt dizzy, became pale, and would have fallen had

he not supported himself by holding on to a railing. This state lasted about a-quarter of an hour.

When admitted, he was listless and drowsy, and expressed himself slowly, but connectedly. Pupils normal and equal. Pulse 96, full, and equal in both wrists; visible pulsation in the radial and ulnar arteries. His aspect was fresh, and there was no oedema. Heart's impulse feeble and not extended. At midsternum a double murmur was heard, of which the diastolic element was the louder; it was transmitted in the course of the aorta, and at the second right costo-sternal articulation the systolic murmur was the louder. Here, likewise, it was coarse and jarring, and was alone transmitted into the carotids, which pulsated visibly but feebly. To have infusion of senega with carb. ammonia, grs. iij for the dose, thrice daily.

The annexed tracing of the pulse was taken by my friend Dr. Grimshaw on the 26th. On the 30th the man was discharged at his own request, much improved in general health, but with the physical signs unaltered.

FIG. XL.



James D.

Weak, but regular pulse; fatty degeneration, with obstruction and regurgitation at the aortic orifice.

There was no opportunity for testing the diagnosis in this case by *post mortem* examination; but I think there can be no doubt it was, in its main features, correct.

#### CASE LV.—*Anæmia, and Fatty Heart; Dilatation of the Aorta.*

Stephen McN., a porter in the butter market, aged thirty-seven years, temperate, presented himself as an extern patient at the Mater Misericordiæ Hospital, May 17th, 1867.

Twelve months previously he began to experience strange sensations in his head, and since that time he has been subject to giddiness, during which he has a tendency to fall down; has never had a fit, or become insensible. He is remarkably pale and waxy, and somewhat bloated in appearance, but not œdematous; the surface is cold, and he is very nervous. Both corneæ are somewhat opaque. Pulse 72, and weak. Respiration ordinarily 36 in the minute, but subject to remarkable variations of rate and character. Thus, the man being at rest, and the respiration 36, and inaudible to a bystander, it is liable to become quick, loud, and panting. In this state it would continue a few minutes, then subside to the previous rate, and resume its former character.

The surface of the chest is soft and puffy, but not œdematous; the heart's action is feeble; first sound scarcely audible; second sound much louder than the first, but less distinct than in the healthy organ; no murmur exists.

This man improved much under treatment with iron and quassia, and after a few visits he ceased to attend.

CASE LVI.—*Recurrent Vertigo; Orthopnœa; Chronic Cough; Rigidity and Tortuosity of Temporal Arteries; Urine slightly Albuminous, but sp. gr. 1·025; Pulse Regular; Hæmoptysis; Jugular Pulsation; Great Œdema and Tension of Lower Limbs, and Genitals; Erysipelatous Inflammation of latter; Left Apex-Systolic Murmur transmitted to Base; Death. Heart Enlarged; Right Chambers Dilated and Thinned, and containing a Band of Decolorized Fibrin, which passed through Tricuspid Orifice, and rendered Valve Incompetent; Tricuspid Opening Dilated; Left Chambers Dilated and Thickened; Dilatation being in excess in the Left Ventricle; Mitral Valve Sound and Competent; Aortic Valve also Healthy and Competent; Aorta Dilated above Orifice, and Atheromatous; Liver, Kidneys, and Lungs Congested; Advanced Fatty Degeneration of the Heart.*

Thomas O., aged sixty-nine years, a float driver, admitted



April 22nd, and placed under my care, May 1st, 1869, for pulmonary emphysema with chronic cough. He has had vertigo on exertion, but he never fainted. There is orthopnœa. Pulse, 108, regular; temporal arteries tortuous and rigid; impulse of heart heaving; both sounds masked and dull, but free from murmur; slight œdema of the feet.

After some weeks' residence, and under tonic treatment, he was discharged in improved health.

June 25th, re-admitted. Lower extremities and genitals are now enormously swollen, and on the left leg are some large red patches of an erysipelatous character. Urine, sp. gr. 1.025, and containing some albumen. Pulse 108, full and regular; cardiac impulse represented by an obscure and diffused heaving movement; no apex-pulsation to be detected, but below the nipple, and to the left of the nipple-line, a soft murmur, replacing the first sound, is audible; second sound weak, but normal. A transmitted systolic murmur is faintly audible at the base. Respiration 36, not embarrassed; slight expectoration of blood-stained mucus; jugular pulsation on right side. To have compound decoction of scopolarium, ʒviiss, with nitrous spirit of ether, ʒiv; an ounce to be taken every second hour.

July 4th. Respiration has been greatly embarrassed during the last few days. Lower limbs swollen and tense, and the genitals in a state of erysipelatous inflammation. The pupils are contracted, and the patient is somnolent. At 12, A.M., he died quietly.

On the following day the body was examined.

The liver, spleen, and lungs were congested, but not otherwise altered. The heart weighed twenty-three ounces; it was soft, and, when laid on the table, it became expanded by its own weight.

The right ventricle was covered with a uniform layer of fat, two lines thick, and on its anterior surface, near the base, there was a large, white, irregular patch, caused by subserous thickening or deposit. The apex was blunted, and formed of the left ventricle exclusively. The aorta, near the heart, was greatly dilated, and atheromatous in patches; but the valves were sound and competent, as dilatation commenced only at the sinuses.

The right auricle and ventricle contained a ribbon-shaped

band of white fibrin ; this band was attached by one extremity to the muscoli pectinati of the auricular appendix, and, passing through the auriculo-ventricular orifice, it fixed the left segment of the tricuspid valve to the anterior margin of the opening ; whilst, by the other extremity, it was attached to the carneæ columnæ of the anterior wall of the ventricle. The right auricle was dilated, and the tricuspid orifice so much dilated that the four fingers and thumb passed through it together. The right ventricle was greatly dilated and thinned ; the left auricle, dilated and thickened ; mitral orifice normal, and mitral valves healthy and competent ; left ventricle greatly dilated, and somewhat hypertrophied. The section of its walls presented the appearance of healthy structure, with the exception of the internal portion, including the fleshy columns, which were of a muddy yellow colour. Under the microscope, this portion of the wall presented the condition of granular degeneration, and here and there lines of oil dots were disposed in the length of the fibre.

In some portions of the field, fibres were presented in the stage of "obsolescence," having lost all appearance of definite structure and become greatly reduced in diameter. Transverse striæ were observable in the remaining portion of the wall of the left ventricle, but granular change had made some progress, and a few large oil dots were visible. The inner layer of the right ventricle consisted of fibres of a similar character to those composing the deep portion of the left ventricle ; they exhibited granular change, with linear series of minute oil dots at intervals, and a few fibres in the state of obsolescence. One of these, where most reduced in diameter, measured the 1,776th part of an inch, and at an adjacent point, where, likewise, all appearance of definite structure had been abolished, the 1,110th part of an inch.

Jugular pulsation was noted nine days before death in this case, and extreme dyspnœa only a few days previous to that event. The inference seems, therefore, warranted, that, of the two causes adequate to the production of tricuspid incompetency, which the autopsy revealed, namely, extreme dilatation of the tricuspid orifice without proportionate expansion of the valves, and entanglement of a segment of the latter by a fibri-

nous thrombus, the former was that which actually gave rise to regurgitation, at least at this early date.

The existence of a murmur of mitral regurgitation, in the absence of lesion, or proportionate inadequacy of the mitral valve, as shown by the autopsy, is to be explained, as in many other examples of the same kind already given, only on the assumption of temporary yielding of the softened and dilated ventricle at the acme of systole, under the centrifugal pressure of the contained blood.

The next case, though observed at a much later date, is so germane to the preceding, that I deem this the most suitable place for its insertion.

CASE LVII.—*Chronic Bronchitis, Anasarca, and Ascites; Arcus Senilis; Rigidity, and Tortuosity of the Arteries; Irregularity of Respiration; Great Œdema and Sloughing of the Genitals; Feeble Cardiac Impulse; Faint and Fugacious Basic Systolic Murmur; Death by Asthenia. Ascites; Cirrhosis of the Liver, and Enlargement of the Spleen; Slight Enlargement of the Kidneys; Enlargement, and Advanced Fatty Degeneration of the Heart; Roots of Aortic Valves Thickened and Rigid, but the Valves otherwise Sound, and Competent; Mitral Valves Competent; Right Chambers Dilated; Left Auricle Dilated; Left Ventricle Thickened, but not Dilated; Arch of Aorta, from above the Sinuses of Valsalva, Dilated and highly Atheromatous, and its Lining Membrane Tinted Bright Red.*

John MacD., aged seventy years, a tailor, had drunk hard in early life, was admitted into the Mater Misericordiæ Hospital, February 6th, 1873. Six years previously, his breathing became oppressed, and obliged him to leave off work. One year later, he had rheumatism in the right shoulder; and five weeks before admittance, his feet began to swell.

When admitted, he was in the following condition. Lower extremities and genitals much swollen; face puffed, and surface generally pallid and pasty looking, except the feet, which were livid. Well marked arcus in both corneæ; abdomen dis-

tended with liquid, but no turgescence of the superficial veins; slight oedema of the thoracic walls; cough with copious frothy expectoration; respiration averaging 30 in the minute, but exhibiting variation of rhythm. Thus, it was occasionally reduced to 24, and then gradually rose to 36, and at intervals of a few minutes it was entirely suspended for a few seconds. Pulse 108, regular, but very feeble, and visible; the right radial artery was rigid, and so tortuous, that about three inches above the wrist it passed to the outer and posterior surface of the radius, and after describing a long and gradual curve, resumed its position in front of that bone, and passed in the ordinary course beneath the extensor tendons of the thumb.

There was throbbing of the carotids. Precordial dulness was nearly abolished; no cardiac impulse to be felt; but at the lower end of the sternum the sounds of the heart were faintly heard. In this situation the first sound was feeble, toneless, and somewhat prolonged; the second sound weak, but rather sharp. A faint soft murmur was heard here, but, owing to the existence of loud bronchial râles, it was not possible to determine its rhythm satisfactorily. Bowels confined. Urine passed in moderate quantity, sp. gr. 1.020, and containing phosphates, but no albumen.

After a few days of treatment by means of stimulant expectorants, dry-cupping, and poultices to the chest, the poor man experienced relief from the bronchitis, and the murmur at lower sternum was determined to be systolic in rhythm; it was exceedingly faint, and not audible in the aorta or in the carotids.

In the further progress of this case, it was noticed that the pulse was occasionally irregular and intermittent; and, at the same time, the murmur was not audible; both events coinciding with an unfavourable change after a bad night's rest, or intolerance of food by the stomach. In the course of a day or two, when improvement had been effected by mild treatment, the murmur would again be heard.

On the 20th, the penis and scrotum being red and tense, were punctured; and on the following day they were passing into a state of gangrene. The features were now collapsed, and the pulse all but imperceptible.



On the 22nd he died of weakness; and on the following day the body was examined by my resident clinical clerk, Mr. Shanahan.

The abdomen was found distended with serum; the liver contracted, and as if seamed on the surface in several places. On section in these situations, the thickened capsule was seen to penetrate the organ. The parenchyma was of a light brick tint, and, under the microscope, the hepatic cells were seen to contain, each, several minute oil dots; free oil was likewise diffused over the field in moderate quantity.

The kidneys were slightly enlarged, weighing five and six ounces respectively. The cortex was thickened, light in colour, and, under the microscope, the tubes were seen full of granular *débris*, and many of them dilated and constricted at intervals; oil dots were likewise seen in the tubes, and free oil dispersed over the field of vision. Many of the tubes were thickened, and presented the concentric arrangement of fibres described and figured by Dr. George Johnson.\*

Lungs congested and lacerable, the right being enlarged. The heart was soft and flabby, and expanded by its own weight when laid upon the table. It weighed thirteen ounces and six drachms.

Aorta dilated, dotted on the internal surface with yellow patches, and in the transverse portion, rough with small rigid plates projecting by their edges from the surface. This condition existed likewise in the roots of the three great branches arising from the arch. The lining membrane was of a deep crimson tint, not removable by washing. The dilatation of the aorta commenced above the sinuses of Valsalva, and did not implicate the orifice. The aortic valves were competent, and structurally healthy, but on the ventricular aspect the festoon-shaped roots projected as thick and rigid seams, and must have given rise to an eddy in the current of exit. Anterior segment of mitral valve, to the extent of one-half its depth from the attached margin, thick and opaque, but not rough or incompetent. Right chambers dilated, but not thinned; the outer surface being covered by a layer of fat one line thick. The right auricle contained a fibrinous thrombus. Tricuspid orifice not dilated;

\* *British Medical Journal*, February 15th, 1873.

tricuspid valves sound. The muscular substance of the right ventricle was of a light fawn tint; and, under the microscope, the fibres exhibited oil dots irregularly dispersed, but crowded in most of them. In a few, the dots existed only in groups in the situation of the nuclei, the greater portion of the surface of these fibres having lost all appearance of definite structure. One of the fibres measured the 888th part of an inch; and another in the same field, but in the stage of obsolescence, only half that diameter, viz., the 1,776th part of an inch. (See engravings, Figs. XXXVIII. and XXXIX., pp. 614 and 615.) The left auricle was thickened, and somewhat dilated; the mitral orifice and valves (with the exception already mentioned) normal; the left ventricle was hypertrophied, but not dilated; its muscular substance was less light in colour than that of the right, but exhibited similar histological changes, except that of obsolescence. No striae were anywhere visible in the structure of either ventricle.

CASE LVIII.—*Fatty Heart and Atheromatous Aorta; Death.*

Rev. Mr. C., aged sixty-five years, tall and corpulent, consulted me on the 14th of July, 1869. He had had repeated attacks of rheumatic gout which had left his finger-joints enlarged and distorted; and, for the last few years, occasional vertigo with temporary loss of vision. Bowels constipated, and some hepatic tenderness. Respiratory sounds and pulmonary percussion resonance normal. Pulse arhythmically irregular and intermittent. Both cardiac sounds obscure below the nipple, and no distinct impulse or apex-beat perceptible. No murmur existed at the apex. At the right base, and for a distance of about two inches in the course of the aorta, a rough systolic murmur was audible, the second sound being clear. No murmur in the carotids, and no cedema. To have, every night, a pill consisting of blue pill, gr. j, and co. rhubarb pill and extract of taraxacum, of each grs. ij. During the day he was directed to take, three times, a table-spoonful of the following: Nitro-hydrochloric acid, ʒij; Liquid extract of taraxacum, ʒiv; Infusion of cascarilla, to ʒxij. M.

July 20th. Pulse 96, regular; kidneys acting imperfectly.

To have, thrice daily, an ounce of the following ; viz., Tincture of colchicum, 3iss ; Spirit of nitrous ether, 3iv ; Camphor water, to 3xii. M. To continue pills, and paint enlarged finger-joints with liniment of iodine.

He returned home to a distant part of the country, and I lost sight of him. I subsequently learned that he died dropsical in January of the following year.

CASE LIX.—*Orthopnœa, Œdema, Hæmoptysis, Albuminuria ; Enlargement of the Liver ; Double Cardiac Impulse ; First Sound Dull ; Second Sound Clear and Sharp ; no Murmur. Diagnosis : Renal Disease (probably Granular) ; Atheroma of the Aorta ; Hypertrophy and Fatty Degeneration of the Heart.*

Rev. Mr. L., aged forty-eight years, consulted me, May 28th, 1872. Had had several attacks of rheumatism ; for a short time preceding March 25th, he had cough, and on that night was suddenly attacked with severe cough and spitting of blood, accompanied by pain in the chest. Under medical advice he now took a few mercurial powders, which salivated him profusely. A week or two previously to his visit to me, his feet began to swell, and his breathing became much embarrassed. For the last few nights he was unable to lie down, and obtained only an occasional snatch of sleep in the stooping posture, resting his elbows on his knees. Within that period also he spat a little blood.

The following was his condition when I first examined him. The features were thin and shrunk ; body plump, but remarkably pale and flabby ; feet and legs greatly swollen. Liver enlarged. Urine acid, in sp. gr. 1·015, slightly albuminous, and containing fatty epithelial *débris*. Pulse 138, small and regular. Cardiac impulse double ; apex-beat half an inch external to the line of the left nipple. First sound masked, and second sound clear and sharp. No cardiac murmur. To have aperient pills. To take, thrice daily, tincture of perchloride of iron, tincture of digitalis, and spirit of chloroform, of each ℥x, in an ounce of water ; and at night a sedative draught of chloral hydrate (grs. xv), and liquor of opium (℥x), in an ounce of water.

May 30th. Slept very little last night, the greater part of which he passed in an arm-chair. Much oedema of feet, and great dyspnœa. Urine increased in quantity. To have mustard cataplasm to chest, and continue medicine.

June 1st. Much improved; quite free from oppression. Slept the greater part of last night in bed. Pulse 84, much more full, and quite regular; breathing tranquil. About three pints of urine were passed within the last eighteen hours. Some blood was passed from the bowels; but he has old hæmorrhoids. I now learned that the patient, having a dread of sedatives, *had not taken the chloral and opium draught till last night.*

4th. Swelling of the lower limbs has in a great degree subsided, and is now confined to the insteps and ankles. Urine averages about two quarts in the twenty-four hours. Can sleep soundly in the recumbent posture, and throughout the night, after his draught. No oppression or acceleration of breathing. Has only very slight and occasional cough, with simple mucous expectoration untinged with blood. Bowels regular; motions unaccompanied by needing, and free from blood. Pulse 84; strong, full, and regular, and presenting the characteristics peculiar to hypertrophy of the left ventricle. Appetite somewhat improved.

6th. Pulse quite regular. Cardiac impulse and sounds regular and distinct. He can now sleep with enjoyment in the recumbent posture, and take solid food; he walked to my house to-day, a distance of half a mile, without inconvenience. Has scarcely any cough, and swelling of feet has nearly disappeared. To have, thrice daily, Sulphate of quinia, gr. iss; Liquor of strychnia, ℥iv, in an ounce of water.

September 20th. Since last report he has had repeated bleeding from the bowels; the feet became again swollen; the right lung congested, and there was circumscribed pleuritis in the left mammary region. The pulse continued steady, and the patient was able to rest in the horizontal posture up to his death, which took place suddenly and quietly at 2, A.M., this morning.

The sudden break up of this gentleman's health after the injudicious administration of mercury deserves special notice. This I believe to have been the result of pre-existing renal



disease. In the combination of granular disease of the kidneys with consecutive hypertrophy and fatty degeneration of the heart, I have found even the smallest quantity of mercury inadmissible. Two grains of calomel have, to my knowledge, caused salivation and great debility in a case of this kind. The improvement in the condition of the patient under treatment with iron and digitalis, and a sedative of chloral and opium at night, likewise deserves notice. The kidneys acted vigorously under this treatment; the œdema all but disappeared; the heart acted with greater force; the breathing and the appetite were improved; sleep was enjoyed in the recumbent posture, and bodily strength was, in a great degree, restored. The improvement was, however, as in all such cases, only temporary. The body was not examined after death; but the diagnosis given in the title of the case, seems, in my judgment, fully warranted by the preceding history.

CASE LX.—*Acute Articular Rheumatism; General Depression; Weak Pulse; Sudden Death. Fatty Degeneration of the Heart.*

Alice M'C., aged about forty years, was admitted into the Mater Misericordiæ Hospital, December 28th, 1871. She suffered from acute articular rheumatism, which dated three days back. The heart was not engaged, and the only noteworthy symptoms exhibited were general depression, with a remarkable weakness of pulse but without irregularity or intermission.

She died quite suddenly on the 3rd of January, 1872, on making a slight movement in bed. The fibres of the heart were found in the so-called "glassy" stage of fatty change.

This case well illustrates the remark previously made as to the gravity of all acute inflammations, no matter how trivial in themselves, where fatty degeneration of the heart exists even in an early stage.

CASE LXI.—*General Dropsy and Venous Congestion ; Dyspnœa ; Slight Albuminuria ; Extension of Precordial Dulness ; Systolic Apex-Murmur ; Transmitted Systolic Basic-Murmur ; Death by Coma. Enlargement of Heart ; Mitral Stenosis ; Narrowing, and Capillary Injection of the Aorta ; Pulmonary Congestion and slight Emphysema ; Granular Degeneration of the Substance of the Heart.*

Mary A., aged twenty-eight years, admitted January 4th, 1873. Had rheumatism when twelve years old, and since then has been delicate, and for the last few years, easily put out of breath. Never spat blood. Three weeks ago the feet and legs began to swell.

When admitted she was suffering from general dropsy, accompanied with great dyspnœa. The lower limbs were livid and greatly swollen ; there was ascites, and the face was puffed, and of a purple tint ; the root of the neck was swollen, and the cervical veins were distended. Pulse scarcely perceptible at the wrists ; but, counted by the heart, it was 84, and regular. Precordial dulness much extended to the right, and the cardiac impulse strong, especially behind the lower sternum. The apex-beat was half an inch external to the nipple-line ; and here a remarkably loud systolic bellows-murmur was audible, but no second sound. At the base there was also a loud bellows-murmur of systolic rhythm, but less loud than the former, and manifestly transmitted from the apex. In the course of the ascending aorta, and over the front of the chest generally, this murmur was likewise audible, but faintly in proportion to the distance from the apex ; it was *not* transmitted into the carotids. At midsternum the second sound was very faint, but somewhat more distinct over the root of the pulmonary artery. Urine passed in small quantity ; it contained a trace of albumen, amorphous sediment of lithate of ammonia, and many octohedral crystals of oxalate of lime.

Under the use of active purgatives and digitalis with iron, she improved, and was able to obtain short sleep in the recumbent posture ; the face lost much of its previous lividity ; the excretion of urine was increased ; the radial pulse became perceptible, was

84, and regular. She was, however, restless at night; and on the 9th, a sedative draught, containing ℞v of the sedative liquor of opium (Battley), was ordered. In the course of the night she became comatose, and died at 3, P.M. the following day despite the most active measures to resuscitate her, including electricity, strong coffee, brandy, hypodermic injection of atropine, and vigorous slapping on the palms and soles. This latter measure alone succeeded in rousing her for a short time; whilst the magneto-electric current, directed from the neck to the precordium, rather depressed the action of the heart.

The body was examined on the 11th January. There were several pints of serum in each pleura. The lungs were congested, and slightly emphysematous on the anterior edges, but otherwise healthy. The pericardium contained about eight ounces of serum. The heart was large, weighing thirteen and a-half ounces, and flaccid on the right side. The posterior surface of the right auricle was studded with granular lymph, the product of recent localized pericarditis; a firm band of lymph connected the anterior surface of the right ventricle, where an opaque patch existed, with the phrenic portion of the pericardium. The right auricle was greatly enlarged, and somewhat thickened, as was likewise the right ventricle; in the latter chamber a yellow flake of fibrin was found impacted between the right segment of the tricuspid valve and the wall of the ventricle. The tricuspid orifice was greatly dilated. The pulmonary artery was likewise dilated, but its valves were healthy and competent. The left auricle was greatly dilated and thickened, and the pulmonary veins dilated. The mitral orifice was reduced to the size of the point of the little finger; it was of a button-hole figure, and smooth and thick on the edges. The papillary muscles were not hypertrophied; but the tendinous chords were thickened, shortened, and fused together. On the ventricular surface, the segments of the mitral valve were seen to form by their union a nearly horizontal diaphragm, slightly bulging towards the ventricle, with a button-hole slit in its centre; the long diameter of this slit was directed from before and from the left, backwards and towards the right side. The left ventricle was somewhat thickened, but its cavity was nor-

mal. The aorta was reduced in size, and its lining membrane was of a deep crimson red colour, not removable by washing. The right and left anterior segments of the aortic valve were thick, opaque, and flaccid; on their ventricular surface, at some distance from the free margin, there were attached two or three nodules of rough lymph, of the size of a pin's head. The valves were, however, competent. Some dark clotted blood was found in the left auricle and ventricle. Under the microscope ( $\times 222$ ), the substance of the right ventricle presented a typical example of the granular stage of fatty degeneration. The fibres had lost, nearly throughout, all appearance of transverse striation; they were pale, averaging the 1,110th part of an inch in diameter, and dotted over with dark points, which effected an arrangement, partly longitudinal and partly transverse, in relation to the fibres.\*

There were four gallons of serum in the abdomen; there was slight adhesion of the convex surface of the liver to the diaphragm; the kidneys were of normal size, but the cortex was thick, opaque, and rather yellow.

For five days this poor woman was under my observation, and during that time I examined the heart daily; yet I did not detect the pathognomonic murmur of mitral narrowing, that of mitral reflux alone having been identified.

This is the only case of mitral obstruction which has come under my notice, in which a sufficient opportunity for stethoscopic exploration having been afforded, the patient being not actually moribund when examined, I have failed to detect a pre-systolic murmur and diagnose therefrom mitral narrowing, proved by dissection to have existed at the time.

I can explain the exception only by supposing that the contractile power of the left auricle was so impaired by effusion into the pericardium and pleural cavities, that it was incapable of propelling its contents with sufficient vigour to produce a murmur.

Fatal coma, in the main, no doubt, due to general serous effusion, was probably aggravated by the moderate dose of opium given.

\* See Fig. XXXVII., p. 613.



That the basic systolic murmur was transmitted from the apex, I infer from the absence of murmur in the carotids, and from the manifest inadequacy of the lesion at the orifice of the aorta, to cause a murmur of efflux.

CASE LXII.—*Pulmonary Emphysema and Chronic Bronchitis ; Recurrent Syncope ; Irregularity of Breathing ; Systolic Murmur at Lower Sternum ; Sudden Death by Syncope. Enlargement of the Heart ; Fatty Growth upon its Surface, and Fatty Degeneration of its Substance ; Dilatation, Atheroma, and Roughening of the Aorta ; Slight Opacity and Thickening of the Aortic Valves.*

Patrick M'E., aged fifty-three years, a gardener, admitted into the Mater Misericordiæ Hospital, January 20th, 1873. He was then suffering from cough and dyspnoea. Two years previously he was in hospital, and under my care, for pleuritis on the right side. A fortnight before the date of admittance, whilst walking in the street, he felt a "glow" all over his body, he became dizzy, and he would have fallen had he not held on by some support within his reach. Since then he has actually fallen in the street and lost consciousness, but has "come to" in the course of a few minutes.

When admitted, he exhibited no œdema, or visible pulsation. The pulse was 84, and scarcely to be felt. There was evidence of pulmonary emphysema with bronchitis. No cardiac impulse to be felt. Respiration varying in rate, even in the recumbent posture, and occasionally suspended for a few seconds.

At the lower third of the sternum, a soft bellows-murmur, regarded at the time as diastolic in rhythm, but really systolic, was heard ; the difficulty of identification arose from the circumstance that the heart being in some degree overlain by the lungs, the cardiac sounds were audible only at distant intervals amid the discord of bronchitic râles. The diagnosis made was, pulmonary emphysema with bronchitis, enlarged and fatty heart, and disease at the orifice of the aorta. To have tincture of digitalis, tincture of perchloride of iron, and spirit of nitrous ether, of each ℥x, thrice daily ; blister and a poultice to chest.

On the 24th he was much improved in regard to the pul-

monary affection, and now the murmur at the lower end of the sternum was readily identified as systolic in rhythm. No second sound was audible in this situation. During the succeeding two days no murmur could be heard ; but on the 27th it was again audible, and recognized as systolic. On the latter date the patient was aphonic ; pulse exceedingly weak ; in the evening he died in one of his usual fits of syncope.

The body was examined next day with the following result :

The lungs were large, emphysematous, and congested posteriorly, and on the right side there was evidence of former pleuritis. Trachea dilated. There were a few ounces of serum in the pericardium. The heart was enlarged, weighing eighteen ounces, and covered at the base and over the right ventricle with a thick layer of fat. In the latter situation the fat extended to a depth of two lines, and somewhat more deeply at the apex. The right chambers were dilated, and full of dark clotted blood. Left auricle normal ; left ventricle slightly dilated and thickened, except at the apex, where it was not more than three lines thick. The valves were all healthy, except those of the aorta, which were opaque and slightly thickened, but competent ; they could not have caused a murmur of exit or of reflux. The aorta was greatly dilated from a few lines above the valves throughout the arch ; it was rough, and fissured through its internal and middle coats in several situations ; and about one inch above the valves, a layer of fibrin, as large as a sixpenny-piece, was firmly attached to one of these rough patches. The muscular fibre of both ventricles was in an early stage of fatty degeneration, exhibiting groups of oil dots in the situation of the nuclei. Elsewhere the fibres were smooth and pale, and in most situations devoid of all trace of striation ; the latter appeared to a very limited extent in the fibres of the left ventricle only, which were of a redder tint than those of the right ventricle.

The murmur heard at the base of the heart was not of valvular origin ; it was caused by the rough state of the interior of the aorta ; and the great dilatation of this vessel, combined with the weak state of the heart, was the cause, at once, of the recurrent syncope, irregularity of breathing, and suppression of the second sound.

CASE LXIII.—*Vertigo, and great Debility ; Dyspnœa ; Weak and Visible Pulsation of the Radial Arteries ; Systolic Basic Murmur Transmitted into the Carotids ; Sharp and Clear Second Sound without Murmur ; Subsequent Suppression of Second Sound ; Acute Pericarditis ; Death. Emphysema and Engorgement of Lungs ; Hypertrophy and Granular Degeneration of Heart ; Villous Deposit of Lymph on the Pericardium and Heart ; Calcareous Transformation of the Aortic Valves ; Dilatation and Atheroma of the Aorta ; Liver, Kidneys, and Spleen Congested, but Structurally Sound.*

Patrick S., aged fifty-five years, a labourer, of temperate habits, was admitted into the Mater Misericordiæ Hospital on the 5th of February, 1872, suffering from cough, dyspnœa, and great debility. He stated that twenty years previously he had rheumatic fever, after which he was subject to winter cold and cough. Five weeks before admittance he began to experience dizziness ; and, three weeks later, great and general weakness. There was, at the date of his admittance, evidence of pulmonary emphysema and sub-acute bronchitis. The respirations were 24 in the minute ; the pulse was 72, soft and weak, but regular ; it was visible in the radial arteries. Precordial dulness was all but abolished ; there was no distinct cardiac impulse, which was represented by a diffused and heaving movement over the lower sternum. The apex was felt to pulsate faintly half an inch below the nipple, and in the nipple-line. At the base of the ensiform cartilage a loud systolic murmur was heard ; it was diffused over the entire front of the chest, and transmitted into the arteries of the neck, louder in the sitting than in the recumbent posture, and loudest at the root of the aorta and in the ascending portion of the arch. The second sound was sharp and clear.

Under a tonic and sustaining plan of treatment there was gradual improvement, and in the course of a few weeks the patient left hospital to resume his work. The cardiac signs, however, remained unaltered.

On the 27th of February, 1873, this poor man was again admitted under my care. He was then suffering from great oppression of breathing, orthopnœa, cough, and extreme weakness.

The sputa were copious and frothy, but uncoloured. Pulse feeble, small, and visible at the wrists. A soft systolic basic murmur existed, as in the first instance; but there was now no second sound to be heard, and no diastolic murmur. Under the use of digitalis and iron, with wine, and counter-irritation to the chest, he improved considerably, was able to lie down, and to sleep in the recumbent posture.

About the 6th of March he was attacked with acute pericarditis; a double and very loud friction-sound was audible all over the precordium, and not modified by respiration; effusion into the pericardium gradually ensued, and he died of asthenia on the 12th of March.

The lungs were found of large volume, emphysematous, congested posteriorly, enveloped in old and dense false membrane, and universally adherent to the chest-walls. The base of the left lung was in the first stage of pneumonia. The pericardium contained about eight ounces of sanguinolent serum, and its internal surface, both parietal and visceral, was studded throughout with villous lymph resembling the dorsum of a sheep's tongue. The heart was globular, enlarged, and, with the pericardium attached, weighed twenty-three and a-half ounces; its substance was fawn-coloured, and in the granular stage of fatty degeneration. The left ventricle was dilated in a slight degree, but greatly thickened, its walls near the base measuring three-quarters of an inch. The right ventricle was dilated and thickened. Both ventricles contained decolorized cylinders of fibrin, extending into the aorta and pulmonary artery respectively. The mitral valve was competent, and, with the exception of the attached margin of the antero-right segment, which was calcified to the extent of about two lines of its depth, perfectly sound. The aortic valves were rolled up in the direction of the aorta, and completely calcified; the posterior-left segment forming with the anterior mitral an uninterrupted calcareous plate. The aperture left by the disorganized aortic valves was in the axis of the opening, and admitted only the point of the little finger. Water poured into the aorta readily flowed into the ventricle through this aperture. The coronary arteries were sound. From above the valves the aorta was dilated throughout



the arch ; it was likewise in some degree rigid and patchy with atheroma, the latter condition extending into the roots of the great arteries of the neck. The liver was large, but otherwise healthy, as were likewise the kidneys and spleen.

I may mention that in January, 1872, the diagnosis made, as recorded in my notes of the case taken at the time, was hypertrophy, obstruction at the orifice of the aorta, and dilatation of that vessel. At no period during his residence in hospital was diastolic murmur audible.

Dr. Stokes has remarked upon the frequent absence of diastolic murmur in aortic patency, where fatty disease of the heart and aortic obstruction likewise exist. I can confirm this observation, and would suggest that the explanation may be found in the rigid and inelastic state of the aorta, which is usually associated with the twofold condition of fatty heart and thickened or calcified aortic valves. In this state, although reflux may take place, giving rise to feeble, collapsing, and visible pulsation of the superficial arteries, it is not accompanied with sufficient force of current to develop a murmur, owing to the want of elastic reaction in the aorta.

Finally, I would remark, that of the entire superficial surface of the heart, the anterior aspect of the apex was alone smooth and free from papillated deposit of lymph. Professor Law, many years ago, observed that this exemption of the apex is of frequent occurrence, owing to the impact of that portion of the heart's surface against the chest-wall in ventricular systole.

CASE LXIV.—*Renal Disease ; Hypertrophy and Fatty Degeneration of the Heart ; Gangrene of the Right Foot and Leg ; Death by Exhaustion.*

Rev. Mr. G., aged about sixty years, was admitted into hospital, May 3rd, 1873. He had been subject for the preceding year to vertigo and threatened syncope, and for the last three weeks there had been cedema of the lower extremities and genitals. Respiration was accompanied by moist râles all over the chest. The pulse was 96, weak, but regular ; precordial dulness extended ; no perceptible cardiac impulse ; first sound masked and faint, and second sound sharp and clear at base ; there were fits

of paroxysmal dyspnœa at distant intervals. Urine 1·020, and after ebullition exhibiting albumen to one-half its quantity.

About the 6th, it became necessary to puncture the feet and legs, in order to prevent the occurrence of inflammation from tension of the integuments. A copious flow of serum followed; nevertheless, two days later, a large bulla appeared above the internal maleolus of the left leg.

On the following day this burst, œdema subsided, and gangrene proceeded no further in that limb; the dorsum of the right foot now exhibited a livid patch, which soon extended to the toes. Large bullæ, filled with amber-coloured serum, now appeared about the ankle joint, and on the following day, along the leg as far as the calf, which was hot and tender, whilst the entire foot was livid, cold, and insensible. He complained of severe pain in the leg, and was troubled with hiccup, which was relieved by the exhibition, every second hour, of a pill composed of grs. ij of dried soda, and ℥j of creasote. He slept pretty soundly, and in the horizontal posture, but with occasional startings.

He died rather suddenly on Monday, May 12th, without having previously exhibited any marked increase of debility. The body was not examined.

In this, as in Case 46, I think the gangrene of the foot and leg was due to local arteritis and thrombosis.

In the form of degeneration known as "brown atrophy," first described by Rokitansky, the heart is wasted, and its muscular structure of a dark ochre tint. A yellow granular pigment is deposited in the muscular fibres, either uniformly, or in fine rows between the fibrillæ and around the nuclei. Whence these pigment particles proceed is not known. This kind of atrophy, which is always general according to Rindfleisch, is most frequently met with in senile marasmus, wasting from inanition, and in the tubercular and cancerous cachexiæ.\*

*Simple softening* of the heart is a condition anatomically allied to fatty degeneration, but pathologically in no respect kindred to it. As in fatty degeneration, there is a change in colour and

\* *Vide* also p. 588.

consistence; but the histological characteristics of that condition are wanting, the origin and association different, the development more rapid, the duration only temporary, and the prospect from treatment much more favourable. I propose to exclude from consideration here the so-called granular softening, which in no respect differs from the early stage of fatty degeneration; and likewise inflammatory softening, which, by a different process, leads to the same condition. By simple softening of the heart, I mean only the condition of that organ so often associated with essential fevers of the typhus type. In this connexion, Laennec described a condition of the heart characterized by a change of colour either to a violet or a dead-leaf hue, great softening and friability, and diffused over the entire heart or confined to the left ventricle. The cardiac impulse is very feeble, and, according to him, both sounds are indistinct, the first being occasionally suppressed.

Laennec vehemently denies the inflammatory origin of febrile softening of the heart insisted on by Bouillaud, and declares that it is peculiar to the grave forms of essential fever, which are characterized by intumescence of the face, a spongy condition of the gums and lips, a fuliginous deposit upon the tongue and teeth, an earthy colour of the skin, inflation of the abdomen, and fetid dejections from the bowels. The pulse, he says, is remarkably quick throughout the fever; and where it is continued at the same rate into convalescence, he would diagnose persistent softening of the heart; an error of judgment subsequently corrected by Dr. Stokes. Laennec further assumed the coexistence, in most cases, of what he described as "a gummy or fishy" state of the muscles of animal life.\*

Zenker, who examined over one hundred bodies of those who had died of typhus at Dresden, in the epidemic of 1858-62, found the voluntary muscles, chiefly the adductors and the recti abdominis, in a state of degeneration in different degrees, and to a variable extent "no less constantly than the characteristic alteration of the mucous membrane of the intestine." He described two forms of degeneration of the voluntary muscles as witnessed by him in this connexion; namely, the "granular" and the

\* *Auscultation Médiate*, tom. ii., p. 532.

“waxy.” The former, as judged by his account of it, differs in no respect from the granular degeneration of the heart already described as constituting a stage of transition into veritable fatty degeneration. The granules are albuminous, and readily soluble in acetic acid. In the “waxy” form the fasciculi are swollen, in appearance homogeneous, and white and glistening like solid wax. This change is due to the deposit within the sarcolemma, which remains unaltered, of “a peculiar protein substance resulting from the transformation of the syntonin,” and differing entirely from the amyloid. This he regards as the more grave form of the two, as it does not admit of the restoration of the normal structure of the muscle. It is likewise the more common of the two forms, having been found in 70 cases out of 79, and of this number, it was in the advanced stage in 36. Under both forms the muscles are brittle, and in many cases actually fissured across the fasciculi.\*

I have not seen changes in the voluntary muscles corresponding to either of these conditions; they are denied by Stokes and other writers of the highest authority.

Laennec regards softening of the heart as essentially due to a disturbance of nutrition.

Louis, a few years later, noticed, very fully, softening of the heart as occurring in typhoid fever. Out of a total of 47 cases, the heart was more or less softened in 23 of those that died between the eighth and twenty-ninth days of illness; and in 15 out of this number, it was flabby, moulding itself like a wet cloth into any shape given to it. It was, moreover, easily torn, dry in section, not enlarged, and in colour it resembled the peel of an onion. Softening of the heart was found in only one-third of the cases which were fatal between the twentieth and thirty-second day of illness. No other muscular organ was affected, and the voluntary muscles were in a healthy condition. The walls of the heart were thin, and its chambers, in extreme cases, contained only a few drops of dark coagulated blood mixed with air. In nearly all, the lining membrane of the aorta was red, either in patches or uniformly; the change of colour extending to the middle coat; it was likewise thick and soft. Louis denies

\* *Archives Générales de Médecine*, Aout et Septembre, 1865 (abstract).



the alleged inflammatory origin of this condition of the heart, regarding it as the result of a special blood-crisis.\*

The next, and the most valuable contribution to our knowledge of this subject was made by Dr. Stokes.† According to this writer, softening of the heart, at least in Ireland, is peculiar to typhus fever. It is usually associated with a dark petechial eruption, sordes, and a peculiar odour from the body. The heart is livid and soft, especially the left side, pitting on pressure, and easily torn, and often infiltrated with "an adhesive gummy secretion." There is no trace of inflammation, and the valves are unaffected. The external one-eighth of the substance of the left ventricle is homogeneous and without the appearance of muscular structure, dark, and resembling in section the cortex of the kidney. These changes are, in a few cases, exhibited only in patches. The right ventricle and the septum ventriculorum are occasionally affected. He has not seen thinning of the parietes, nor a dry state of the cut surface, as described by Louis. He maintains that in the production of this remarkable change in the structure of the heart inflammation has no share, and adds, "All the anatomical and vital phenomena of this affection point to the opinion that it is an example of one of the special secondary lesions of typhus, like the infiltration of the mucous glands of the intestine, and capable of retrocession, without consequent disorganization."‡ The signs characteristic of this change in the heart commence about the sixth day of illness and cease about the fourteenth. They continue therefore, ordinarily, about eight days. The signs bear intrinsic evidence of debility of the heart, and are briefly as follows: Feebleness, or entire cessation of impulse, and partial or complete suppression of the first sound; coincidently with these modifications of the cardiac phenomena proper, the radial pulse is weak, but regular and rapid; the rate amounting, in many cases, to 150 in the minute. It occasionally happens, that whilst no

\* *Recherches sur la Maladie connue sous le Nom de Gastro-Entérite*, 1829, tom. i., chapitre viii.

† *Dublin Medical Journal*, vol. xv., 1838; and *The Diseases of the Heart and Aorta*, 1854, p. 366.

‡ *Opus citat.*, p. 373.

impulse or first sound can be detected at the apex, both are perceptible over the right ventricle at the lower end of the sternum. Again, whilst no impulse is perceptible in the position of dorsal recumbency, a faint apex-beat may be occasionally felt in the normal position, or somewhat to the left, by causing the patient to turn over on the left side.

Dr. Stokes remarks that in some grave cases he has known the two sounds to be nearly identical in quality, and closely resembling a weak second sound, the action of the heart being at the same time very rapid. This he very appropriately designates "foetal action." Under the free use of wine, and likewise in convalescence, the pulse usually becomes slow, and the first sound again audible; whilst the cutaneous circulation is more active, and the temperature of the extremities rises. These are signs of favourable augury, and may be regarded as evidence of the beneficial action of wine when they follow its administration.

When, on the contrary, between the eighth and the fourteenth day of illness, the action of the heart continues rapid despite an ample allowance of wine, the conclusion may be drawn that stimulants are not agreeing with the patient, or acting unfavourably. If the pulse continue rapid in convalescence, he would infer the presence of some acute inflammatory complication in the chest or abdomen, or phlegmasia dolens.\*

The distinction between softened and simply weak heart rests upon the following characteristics: The latter is due to some constitutional debility of a very palpable character, to a shock, or violent revulsion of some kind. The signs are proportionately rapid in development, and, in the event of a favourable issue, equally prompt in disappearing. The signs of cardiac softening, on the contrary, progress up to a maximum point and then recede, occupying a period of several days; they appear with quick pulse, the impulse of the heart becoming less and less distinct, and failing first at the apex. There is usually, but not always, a corresponding loss of the first sound. In convalescence the pulse may descend to 30 in the minute, and again rise to the natural standard, at which it continues. The impulse is reestab-

\* *Lectures on Fever*, 1874, p. 188-9.

lished before the first sound, and both are perceptible at the ensiform cartilage earlier than at the apex. The observations of Dr. Stokes, epitomized in the foregoing remarks, have advanced considerably the scientific therapeutics of fever. For a full discussion of the subject, which could not be entered upon here, the reader is referred to Dr. Stokes' writings already quoted.

Whether any change in the elementary structure of the heart takes place in simple softening, I am not in a position to declare from personal observation; but that such is not the case, is no more than a legitimate inference from the fact, that within a period of a few weeks complete restoration to health not unfrequently takes place in cases characterized by the above mentioned cardiac phenomena; the action and sounds of the heart resuming, at the same time, their normal characters.

Doctor Murchison, however, in the first edition of his great work on fever, published in 1862, affirmed that in typhus softening of the heart, the muscular structure of the organ is in a state of granular degeneration.

I have not met with the changes in the voluntary muscles described by Zenker; and from his allusion to the state of the intestinal mucous membrane, I conclude that the fever with which he found them associated was what in this country would be regarded as typhoid or enteric. Yet in this form of fever Louis declared that the voluntary muscles, as examined by him, were unaltered. It must, however, be remarked that the means for examination of structure at Louis' disposal were very defective. The subject needs further investigation.

Doctor Ormerod has studied this pathological change in connexion with delirium tremens. He says: "Of five dissections after death from simple uncomplicated delirium tremens, of which I have records, the heart is noticed to have been particularly soft and flabby in four, and in four to have presented numerous small ecchymoses beneath the investing or lining membrane. In one, the heart, though flabby, was not dilated like the rest, but small and loaded with fat. Examination by the microscope failed, however, to detect the slightest evidence of fatty degeneration of its structure."\*

\* *London Medical Gazette*, 1844, p. 792.

In delirium tremens I have occasionally found the heart acting with great feebleness and rapidity, the first sound being all but suppressed. Such a condition involves the utmost danger to life; even a slight effort on the part of the patient may cause syncope and sudden death: an accident which I once witnessed in the case of a professional man well known in Dublin. In such cases I should advise the use of digitalis and iron in large doses.

*Fibroid transformation* of the heart, incidentally noticed at p. 510, is most frequently the result of antecedent inflammation commencing in the pericardium or endocardium and thence extending into the substance of the organ. Inflammation leads to hypertrophy, by causing irritation and actual growth of the connective tissue of the heart, and proportionate absorption of the proper muscular structure. Fibroid change may, however, originate in inflammation of the connective tissue of the myocardium itself; but this is very rare in comparison with the first mentioned mode of origin. Sir W. Jenner maintains that congestion of the substance of the heart may give rise to a modified form of fibrous transformation if slow in occurrence, moderate in degree, and either permanent, or often repeated. In such circumstances, induration, roughening, and thickening of the walls of the heart by interstitial exudation of fibrin may take place; the walls would be firm, tough, and apparently homogeneous on section; they would not, though thin, collapse, and the fleshy columns would stand out. Hence, the cavities would be permanently dilated. The colour may be paler or darker than normal. Microscopically, the structure would be characterized by indistinctness of striation, and close aggregation of fibres, protein molecules being interspersed between them, and in their substance. These molecules are liable to fatty degeneration, and when this change has taken place the previously rigid structure is soft and brittle.\*

I think the condition here indicated is due to sub-acute inflammation of the connective tissue of the heart, and differs only in degree from that about to be described.

Doctor H. C. Wood, of Philadelphia, declares, as the result of

\* *Medico-Chirurgical Transactions*, vol. xliii., p. 199.



numerous experiments in the production of "thermic fever," a form of sun-stroke, which, according to him, does not require for its development actual exposure to the sun, that "excessive rigidity of the heart, by coagulation of its myosin, is pathognomonic of sun-stroke," and usually occurs directly after death.\*

One of the earliest recorded examples of fibroid conversion of the heart was published by Dr. Fothergill.† A gentleman, aged sixty-three, short and full, and subject for three years to fits of angina pectoris, died suddenly in a paroxysm of anger. The body was examined by John Hunter, who reported as follows: "The heart, to external appearance, was sound; but, upon examination, I found that its substance was paler than common, more of a ligamentous consistence, and in many parts of the left ventricle it was become almost white and hard, having just the appearance of a beginning ossification."

Fibroid metamorphosis of the heart is a veritable "connective tissue-hypertrophy," or hyperplasia, and due, as Quain declares, to chronic interstitial inflammation. The inflammatory irritation to which it owes its origin may commence on the surface, external or internal, and thence extend by continuity of texture into the substance of the heart. Pericarditis and endocarditis are, therefore, not infrequent causes of hypertrophy of the connective tissue, or so-called "fibroid" degeneration of the heart.

When the pericardium is adherent, the heart, according to Quain, is generally enlarged, but the increase consists of connective tissue only. When atrophy of the heart coincides with adhesion of the pericardium, there is, he thinks, veritable cirrhosis of the heart: a change which he regards as due to the infiltration of a denser fibroid tissue.‡

I believe, however, that when the substance of the heart has become the seat of hypertrophy of the connective tissue, the volume of the organ may be taken as an index of the age of the hypertrophied structure. Where the heart is large it will be found still to consist in great part of muscular fibre, and the history of the case will show that the antecedent inflammation

\* Prize Essay, reviewed in *Medical Times and Gazette*, October 19th, 1872.

† *Medical Observations and Inquiries*, vol. v., p. 252, 1774.

‡ Lumleian Lectures, *Lancet*, March 23rd, 1872.

has not been very remote. Where, on the other hand, atrophy of the heart has taken place, the muscular fibre has entirely, or all but entirely, disappeared; the remaining fibres, when any such are visible, being in the granular stage of fatty degeneration, as shown by Bristowe.\*

Wilks reports a case in which one-half the muscular substance of the heart was replaced by fibrous tissue, which was in continuity with an adherent pericardium.†

Doctor Bence Jones has recorded a case strikingly illustrative of the causal connexion between surface inflammation of the heart and fibroid change of the subjacent structure. Under the endocardium covering the septum ventriculorum there was a layer of fibroid tissue "only where the endocardium had been inflamed." Mr. Gray, who examined this fibroid material, reports that it consisted "of granules, with nuclei and nucleated cells embedded in it."‡

In another case, Dr. Bristowe reports on a similar fibroid substance found in small detached masses in the substance of the heart, that it consisted "of fibro-plastic deposit, composed of large nucleated cells, granular cells, and nucleated spindle cells, with granular and oily matter, and pervaded by the muscular fibres."§

Rindfleisch urges that parietal endocarditis differs in its results from inflammation of the valves, in the circumstance that it leads to depression or pitting of the surface, thinning and bulging outwards of the wall, and thus to partial aneurism of the heart. The part so affected is small in extent, situate usually on the anterior wall of the left ventricle near the apex, and composed of tendinous structure invested by the pericardium. At the edges a few muscular fibres are still visible, but intermingled with the fibrous structure. This process of tendinous conversion must, in his opinion, be regarded "as a direct extension of the chronic inflammation of the endocardium to the sub-endocardial and intermuscular connective tissue."||

Doctor Lionel Beale regards fibrous transformation of muscle

\* *Transactions of London Pathological Society*, vol. vi.

† *Ibid.*, vol. viii.

‡ *Ibid.*, vol. vii.

§ *Ibid.*

|| *Pathological Histology*, New Sydenham Society's edition, 1872, vol. i., p. 293.

as a veritable degeneration, arising either from old age or from long continued suspension of muscular contraction, as in cases of nerve-paralysis. "In old age," he says, "much of the muscular tissue is replaced by fibrous material." And again: "The contractile material of the muscle has degenerated into fibrous tissue." "Although in the muscles of the aged there is a relative excess of fibrous tissue, it is never, within the limits of health, present in such proportion as to constitute fibroid degeneration."\*

Doctor Ormerod looks upon this change in the heart as one of conservative substitution designed to fortify the walls of an attenuated and weak heart. But where, as in Corvisart's case, the walls of the heart are actually thickened, this view would not be admissible; and that it is not a means ordinarily adopted to strengthen a simply weak heart, is proved by its absence in fatty softening.

I regard fibroid conversion of the heart, whether local or general, as in all cases the result of antecedent inflammation of the connective tissue of the organ. Most frequently the sub-endocardial, or sub-pericardial connective tissue is primarily affected, by extension from the adjacent serous surface, the connective tissue of the myocardium being subsequently involved through continuity of structure. The latter may, however, be primarily engaged. The subsequent changes are those of shrinking and condensation of the neoplastic material, with wasting and ultimate removal of the proper muscular structure by the disturbance of nutrition arising from extrinsic pressure.

Corvisart† gives a remarkable example of rigidity of the heart, caused, no doubt, by fibroid substitution: a problem impossible of solution at that time, owing to the want of microscopic appliances. The patient, a woman of fifty-five, came under his notice in the year 1800. The heart was large; the ventricles rigid and elastic, and, when compressed, sprang back on the removal of pressure to the state of dilatation, like an india-rubber bottle. The left ventricle, when percussed, sounded like a plate of horn. He remarks that this condition, when primary, precludes dilata-

\* *Medical Times and Gazette*, March 20th, 1869.

† Hebb's translation, p. 146.

tion of the affected chambers; and that dilatation, when associated with it, is of anterior date. Quain confirms this observation.

Doctor Ormerod has met with examples of fibroid change so complete, that the heart, without having undergone change of colour, presented a *resemblance* to a leather flask, and was apparently incapable of *contracting*, save very imperfectly.\*

The symptoms to which this change gives rise, even when partial aneurism of the heart results from it, are not characteristic, inasmuch as they have reference rather to the associated lesions of the aorta and coronary arteries, which are usually rigid and calcareous.

Angina pectoris is frequently a prominent symptom, as exemplified in Fothergill's case previously mentioned. The action of the heart and the pulse are irregular, but not constantly, or in all cases; the impulse is partially or entirely suppressed. There is occasionally palpitation, and the cardiac sounds are weak, but curt and sharp.† The "creaking" sounds, assumed theoretically to result from this condition of the heart, I have not heard. In one instance (Case 65) I have noted a regurgitant murmur from inadequacy of the tricuspid valve, consequent upon permanent dilatation of the right ventricle and fibroid transformation of the right papillary muscles; in another case, under the care of my colleague, Dr. Nixon, which he kindly invited me to examine, there was likewise a murmur of regurgitation both at the mitral and the tricuspid orifice. On examination of the body, this was found to be due to fibroid transformation of the inner portion of both ventricular walls and of the papillary muscles, one of which, in the left ventricle, was remarkably attenuated, rigid, and biventral.

In the dissecting room at the School of Medicine, Cecilia-street, a still better example of fibroid transformation of the heart was presented to me in the session of 1873-4, by Mr. Foley, one of the students engaged in dissection. It was obtained from the body of a male subject, aged about forty-five,

\* *British Medical Journal*, 1862.

† Laennec declares, on theoretical grounds, that they are ringing in quality, and heard at a great distance; but such, in fact, is not the case.



regarding whose history or last illness unfortunately nothing whatever could be learned. The heart was somewhat enlarged, the mitral orifice was greatly contracted, and the mitral valves and chordæ tendineæ remarkably thick and rigid. The left papillary muscles were entirely converted into fibrous tissue, and the walls of the left ventricle, to the depth of two-thirds of their thickness, measured from the internal surface, were changed into dense fibroid tissue which was disposed in separate patches, and was continuous with the papillary muscles at their parietal attachment. The endocardium, in the left chambers, was universally thick and opaque.

Save in the early stages, when inflammatory changes are still in progress, nothing can be accomplished curatively. When the process of atrophy has advanced beyond the initiatory stages, the treatment should consist in measures to promote general nutrition and give tone and strength to the heart. Quinine, in combination with strychnia, and digitalis with iron, are the medicinal agents chiefly to be selected. Nutritious diet and alcoholic stimulants in moderate quantity should be allowed, and rapid movement and fatiguing exercise must be forbidden. The following case affords a good example of this variety of transformation.

CASE LXV.—*Pulmonary Emphysema with Recurrent Bronchitis ; Venous Congestion and General Dropsy ; Systolic Murmur at the Ensiform Cartilage and somewhat to the Left ; Death. Enlargement of the Heart ; Hypertrophy and Permanent Dilatation of the Right Ventricle, with Sclerosis of the Right Papillary Muscles, and consequent Inadequacy of the Tricuspid Valve ; Left Ventricle Normal ; Mitral Valve Opaque, but Adequate ; Aortic Valve Healthy.*

Mrs. S., aged thirty-nine years, and the mother of nine children, was admitted for the second time into the Mater Misericordiæ Hospital under my care, on the 7th of January, 1874. Just one year previously she was under treatment in hospital for bronchitis, to which she had been subject in winter for the preceding six or seven years. She was then cupped, and got well in three weeks.

When admitted for the second time she was livid ; the feet

were swollen ; respiration was laboured, and expiration greatly prolonged ; no pulse could be detected at the wrist. The heart pulsated regularly but feebly behind the xiphoid cartilage, and both in this situation and for a distance of an inch and a half to the left, a soft systolic murmur was heard. This murmur was confined within the limits just mentioned, and was accompanied by a faint first sound ; it subsequently assumed a musical character for three or four days, and, at a later period, it was entirely suppressed for about two days. The lower limbs became greatly swollen and required to be punctured. The feet became erysipelatous and ultimately gangrenous. She derived great benefit from large doses of spirits of turpentine and tincture of digitalis ; also from a night-draught containing chloral hydrate and bromide of potassium, of each grs. xx.

She died, worn out, on the 13th of February.

The heart weighed ten ounces. The right ventricle was permanently dilated and much thickened ; its papillary muscles and inner stratum were rigid and firm, and, under the microscope, exhibited fibrous tissue pervading the muscular structure, in the form of broad bands, whilst the muscular fibres were reduced in size, and presented very faint striæ. The tricuspid valve was healthy in structure, but inadequate to cover the orifice, which was dilated proportionately to the ventricle. The left ventricle was normal. The mitral valve was remarkably opaque and somewhat thick, but pliant, and competent to close the orifice. The aortic valves were sound. The surface of the heart was smooth, but presented a large "milk spot" which occupied the usual situation on the anterior surface of the right ventricle. The latter chamber was occupied by clot and some flakes of fibrin. The pericardium contained about eight ounces of serum.

Metamorphosis of the walls of the heart, to a greater or less extent into *cartilage*, is the result of a more advanced stage of fibroid conversion. Professor R. Smith has recorded a good example of this condition ; the apex of the left ventricle, and some of the columnæ corneæ had been converted into dense white cartilage.\* When referring to cartilaginous alteration in con-

\* *Dublin Medical Journal*, vol. ix.

nexion with hypertrophy (p. 510), I stated that I had not met with an example of the kind. Case XL. (p. 592) subsequently came under my notice. In that instance the cartilaginous formation consisted of plates, about one and a-half inch in diameter, ovoid in outline, and about three lines thick; they were embedded in the superficial surface of the heart to which the pericardium closely adhered, and had their origin in antecedent pericarditis.

*Cancer of the heart* is, in the great majority of instances, secondary or metastatic. Rindfleisch asserts that it is never primary, and Peacock could find only two recorded examples. Most frequently the seat of the primary cancer has been the lungs, pleuræ, or anterior mediastinum; its form has been in most cases medullary, with or without melanotic pigmentation.

Doctor Peacock gives a list of 44 cases pretty fully reported by other observers, and 2 which had come under his personal notice.\*

Laennec had seen 2 examples of encephaloid in the heart, and Recamier had mentioned to him 1 of scirrhus.

In a special memoir (*Revue Médicale*, 1824) Andral and Bayle refer to 3 cases which they had seen, and Bouillaud, in his first edition (1835), to 14 cases.

Dr. Peacock's 2 cases were briefly as follows:

Case 1. A young man, aged eighteen years, emaciated, had cough and hæmoptysis; action of heart weak and quick, but no murmur. Extensive infiltration of encephaloid cancer into both lungs and bronchial glands was found. The base of the heart was encircled by a ring of cancer one inch thick, and embedded in the false membrane of an adherent pericardium. The myocardium was unaffected. The liver, the spleen, the mesenteric and Peyer's glands, were cancerous.

Case 2. A young woman of nineteen years; six months ill with cough, hæmoptysis, dyspnœa, and wasting. The left lung was found extensively infiltrated with soft cancer. Two masses projected into the pericardium, penetrated the auricles, and pressed on the vena cava. There was some cancer in the mesenteric glands.

\* *Proceedings of the London Pathological Society*, vol. xvi., p. 100.

Doctor Peacock classifies, under four heads, the cases of cancer of the heart included in his list.

1. Primary cancer of the heart, of which he could find recorded only 2 examples.
2. Heart affected in conjunction with other, and especially with adjacent organs; viz., lungs, pleuræ, mediastinal and bronchial glands: 5 cases.
3. Cancer spreading to the heart, by contiguity, from the mediastinal, cervical, and bronchial glands: 8 examples.
4. Cancer of heart secondary to that of some other organ. This is the most common form, and of it 22 examples are given. They were distributed as follows:

In 3 cases it was primary in the eye;

1 in the cheek and face;

1 „ lower lip;

2 „ breast and axillary glands;

1 „ radius;

1 „ ribs and pleura;

7 „ abdominal organs;

1 „ inguinal glands;

1 „ uterus and vagina;

1 „ labia and clitoris;

1 „ glans penis and inguinal glands;

1 „ testicle;

1 „ thigh.\*

Doctor Risdon Bennett mentions a very remarkable case, communicated to him by Dr. Sutton, in which a primary scirrhus of the left female breast extended through an intercostal space, by a narrow pedicle, to the heart, which it extensively involved.†

Examples of malignant disease have been recorded by Drs. J. Sims,‡ R. Bright,§ Prescott Hewett, and E. L. Ormerod.||

Since the publication of Dr. Peacock's statistics, I find recorded 9 examples of cancer of the heart, which are summarized in the following Table:

\* *Loco citat.*

† *Thoracic Growths*, 1872, p. 144.

‡ *Medico-Chirurgical Transactions*, vol. xviii.

§ *Ibid.*, vol. xxii.

|| *Ibid.*, vol. xxx.



TABLE VIII.—CANCER OF THE HEART.

No.	Author.	Age.	Sex.	Species of Cancer.	Primary.	Secondary.	Penetrated.	Where situated.
1	Dr. Flint	25	M.	Scirrhus	1	...	...	Enveloped heart.
2	Dr. Sutton	53	F.	Scirrhus	...	To scirrhus of left mamma	Right ventricle	In wall of left ventricle.
3	Mr. Bricheteau	24	F.	Scirrhus	...	To scirrhus of ovary	Right ventricle and auricle	Over base of heart.
4	Dr. A. Clark	...	...	Melanotic	...	To melanotic cancer of nearly all the organs	—	—
5	Dr. Stokes	23	M.	Scirrhus and colloid	...	To scirrhus and colloid of posterior mediastinum, and nearly all the abdominal organs	...	Exterior of right auricle and ventricle.
6	Mr. Perssé White	56	F.	Encephaloid	...	To encephaloid of right lung and posterior mediastinum	Pericardium	Exterior of heart.
7	Dr. Ed. Bennett	60	F.	Scirrhus	...	Anterior mediastinum and left lung	Right ventricle	Walls of aorta and pulmonary artery.
8	Dr. Hayden	29	M.	Scirrhus	...	To scirrhus of anterior mediastinum	Pulmonary artery	Embraced aorta and pulmonary artery.
9	Dr. Mayne	45	F.	Scirrhus	...	To scirrhus of anterior mediastinum and left lung	...	Pressing on superior vena cava.

The only example of carcinoma of the heart which I have seen, namely, that included in the preceding Table, was as follows :

CASE LXVI.—*Carcinoma of the Anterior Mediastinum, involving the Aorta, Cervical Vessels, and Pulmonary Artery ; causing Suppression of Carotid Pulsation, Systolic Murmur in the Pulmonary Artery, and Displacement of the Heart Upwards and to the Left ; engaging likewise the Root of the Right Lung, and Compressing the Right Branch of the Pulmonary Artery. Death by Asphyxia from Effusion into both Pleural Cavities.*

Andrew M., aged twenty-nine years, a cabinet-maker, formerly intemperate, but latterly reformed, was seen by me as an extern patient on the 25th July, 1871 ; he was supposed at that time to be suffering from thoracic aneurism. He gave the following account of his illness. Two months previously he got out of health, the first change noticed being a swelling at the root of the neck. He subsequently lost his voice at intervals, and a few days before I saw him, he spat some blood.

When examined by me he was chilled and weak, and could not lie comfortably on the right side. When he attempted to do so he was instantly obliged to turn over, owing to the tumultuous action of the heart which was thereby caused. His face was livid, and especially so the lips, lobes of the ears, and tip of the nose. The neck was tumid, the cervical veins greatly distended, and the upper part of the thorax oedematous and livid. The pulse was 120, very weak, but regular, and somewhat weaker on the right side than on the left. He had no difficulty in swallowing.

A general examination of the chest led to the following results. The entire surface was resonant on percussion, and respiration was audible on both sides at all points, save under the right clavicle, where there was a total absence of respiratory sound. At midsternum there was a harsh murmur, accompanying the first sound of the heart, which was diffused over the entire surface of the sternum ; and over the lower end of the sternum a strong diffused impulse was felt, whilst no movement

was discoverable in the normal seat of cardiac impulse. Systolic murmur was likewise audible on both sides posteriorly, but louder on the right side, internally to the angle of the scapula, than on the left. No impulse was anywhere discoverable posteriorly.

He was admitted into hospital on the 20th September. There was then greater cedema and lividity of face, greater tumefaction of the neck, and distention of the cervical and superficial thoracic veins. There was likewise cedema, but not lividity, of the lower extremities and external genitals. The pulse was 120 and regular, and nearly, but equally, suppressed in both radial arteries. No pulsation was to be felt in the arteries of the neck; but in the carotids a very faint systolic sound was audible, showing that a feeble circulation was still carried on through it. There was no dysphagia. The heart was displaced vertically upwards to the left third intercostal space, where the apex was felt distinctly to pulsate. In this situation, and over the entire anterior surface of the chest, a systolic bellows-murmur was heard; but its point of greatest intensity was in the left second intercostal space, close to the sternum. The second sound was normal. The right front of the chest was comparatively dull; respiratory sound was nearly abolished on this side, especially over the lower two-thirds, and vocal fremitus was absent to the same extent; it was perceptible, however, beneath the clavicle. On the left side dulness likewise existed, except superiorly, and vocal fremitus was distinct only in the subclavicular region. Respiration was audible on both sides posteriorly, but less distinctly on the right, whilst comparative dulness existed on both sides.

Towards the end of September he became subject to occasional paroxysms of dyspnoea at night. The face, neck, and chest became tumid and in the last degree livid, and the right hand and arm much swollen, but decubitus was dexter.

He died quietly on the night of the 7th October; cedema having previously all but disappeared from the genitals and lower limbs.

In July, and the early part of September, I inclined to the view that aneurism existed; subsequent observation led me to

change my opinion, and for the following reasons. First, the dulness in front was not associated with a corresponding impulse. Second, there was *upward* displacement of the heart, and the murmur heard was most distinctly audible *above* the point of apex-pulsation. Third, pulsation in the cervical vessels was completely abolished on both sides: a circumstance scarcely compatible with the existence of aneurism in the situation indicated by the physical signs. Fourth, even if the latter supposition were admissible, an aneurism so situated could scarcely be unaccompanied by tracheal stridor and dysphagia. For all these reasons the alternative diagnosis, that of carcinoma, was made.

On dissection of the body both pleural cavities were found full of serum; the anterior mediastinum was entirely occupied by a scirrhus mass which extended to the ensiform cartilage, penetrated the pericardium, and surrounded the orifices of the aorta and pulmonary artery; it had completely embraced these vessels, and all but occluded the latter. It had displaced the heart upwards, backwards, and to the left, and projected by two nodular growths into the pulmonary artery a short distance above the valves, completely closing the right branch of that vessel, and so constricting the left branch that it barely admitted a director. The arch of the aorta, from an inch above the valves to the descending portion, was completely ensheathed in the solid scirrhus mass, and reduced to the size of an artery of the second order. Its lining membrane was rugose. The innominate and its branches, and the left carotid and subclavian, were likewise surrounded, and were reduced by compression to the size of the brachial artery. The heart was somewhat hypertrophied; it weighed eleven ounces; it was otherwise perfectly sound. Both lungs were considerably engorged, but structurally healthy with one exception; namely, the root of the right lung was contracted and corrugated, being involved in an extension of the scirrhus tumor from the anterior mediastinum. The œsophagus and trachea were not involved. The murmur heard in the left second intercostal space was a veritable systolic murmur in the pulmonary artery, due to the penetration and ingrowth of the cancerous mass.



The annexed engraving, from a coloured sketch taken shortly after death by Mr. Burnside, represents faithfully the condition of the heart and great arteries. Bristles are passed from the arch of the aorta through its primary branches which are embraced by the scirrhus mass. The right ventricle and pulmonary artery are laid open, and the nodular projections of the cancerous tumor into that vessel, by which it was all but completely closed, are exhibited.

The symptoms and signs proper to cancerous tumor implicating the heart are of a vague and indefinite character, having refer-

FIG. XLI.



Cancer of the heart and the anterior mediastinum.

Andrew M.

ence to excentric pressure upon the heart and great vessels, but not indicative of the specific nature of the detruing body. The differential diagnosis will lie between cancerous tumor, and aneurism of the primary portion of the aorta. Thus, displacement of the heart, and the signs of pressure upon the pulmonary artery or superior cava, may be due to either ; as, likewise, dulness on percussion, superficial pulsation, and pain. But displacement by aneurism of the arch of the aorta is never *upwards*, and the signs of pressure are variable in degree and in direction, and more rapid in progress. Absorption of the osseous parietes of the thorax, bulging, and external tumor, are peculiar to aneurism, and positively exclusive of mediastinal cancer ; and, finally, the pulsation of aneurism is heaving, like that of a second cardiac centre. Both are capable of projecting by absorption into the cavities of the heart and great vessels, and of producing identical auscultatory signs ; but the collateral results of such ingrowth differ in the two cases. In the case of aneurism, the walls of the sac having been previously incorporated with those of the heart, pulmonary artery, or superior cava, communication with the interior implies irruption of its contents and a corresponding derangement of the general or pulmonary circulation ; whereas, the ingrowth of cancer involves results having reference only to local obstruction and auscultatory signs. Cancer is slower in progress, more frequently associated with hæmoptysis and signs of pleural and pulmonary inflammation ; usually, likewise, cancer is discoverable elsewhere. The history of aneurism is frequently connected with a definite occurrence, such as pain after a blow upon the chest, or after a strain ; its progress is usually rapid and fitful, and unassociated with disease of other organs except atheromatous degeneration of the arteries.

*Tubercular* development in the heart has been described ; this I have not seen. Todd doubts its occurrence, and Otto has not witnessed it. Miliary tubercle in the pericardium, and acute tubercular pericarditis in connexion with acute tuberculosis of one or both lungs, I have met with. Scrofulous or caseous masses in the substance of the heart have been witnessed by Elliotson\* and by Todd.†

\* *Lumleian Lectures*, p. 32. † *Cyclopædia of Anat. and Physiology*, vol. ii., p. 637.

Townsend describes an example of the same kind. A man aged sixty-four years, was subject for several years to cough and dyspnœa of a remittent character aggravated by damp weather, and, latterly, to paroxysms of dyspnœa threatening suffocation at night. On examination of the body after death, a large quantity of blood was found extravasated in the left lung. The pulmonary veins, especially those of the left side, were greatly dilated, and at the auricular extremity they were reduced to the size of a mole by the pressure of a large "solid mass of tuberculous matter."\*

*Hydatids* have been found in the heart. The cysticercus has been seen in the human heart by Andral; it is often witnessed in that of "measly" pigs.

The late Mr. O'Ferrall exhibited before the Pathological Society of Dublin an example of the same kind. The patient was ill only three months. Tubercles were found in the lungs, and six or seven cysticerci in the septum ventriculorum. There was also chronic renal disease.

Doctor Peacock reported an example of hydatids in the heart, apparently primary, as none were found in the liver or in any other organ.†

I have not met with an example of hydatids in the heart. I should say the symptoms and signs would be most indefinite and misleading.

*Syphiloma* of the heart is very rare, and never met with except in subjects saturated with the syphilitic poison. It belongs to the category of "tertiary" symptoms.

Ricord gives examples of what he regards as syphilitic nodules in the substance of the heart; they were yellow, firm, and solid like cartilage.‡ Other organs, viz., the liver, lungs, spleen, kidneys, and brain, are usually affected in the same subjects.

Lancereaux gives two examples of syphilitic or gummatous myocarditis. The gummata were of the size of peas or cherries, and of a greyish or yellowish hue. The adjacent structure of

\* *Dublin Journal*, vol. i. (old series), May, 1832.

† *London Pathological Society*, October 15th, 1872.

‡ *Clinique Iconograph. de l'Hôpital des Vénéériens*.



the heart resembled tissue undergoing amyloid degeneration. According to Oppolzer, gummata may soften and burst into the chambers of the heart, thereby causing general infection.

Doctor John Morgan has published some interesting cases of the same kind. In one of these, that of a female, aged thirty-seven years, and the subject of chronic syphilis, several cream-coloured gummata were found in the substance of the left ventricle. In all these cases the heart was atrophied, the adult organ weighing in one instance only four ounces, and the impulse and the first sound were feeble. There was likewise, in some instances, an anæmic basic murmur; but no sign of a specific or pathognomonic character existed.\*

According to Dr. Moxon, the elementary structure of syphiloma presents no characteristic feature, consisting only of imperfect and perishing corpuscles, not distinguishable from those of tubercular matter.†

Staff-Surgeon Welsh maintains that syphiloma of the aorta, in the form of so-called atheromatous degeneration, is, amongst soldiers, "the most frequent lesion occasioned by syphilis," and he would explain by this fact the prevalence of aneurism in the army. It was exhibited in the proportion of 57 per cent. of the forty examples of secondary syphilis recorded by him.‡

A case very suggestive of the causal relationship mentioned by Surgeon Welsh has been published by Dr. G. W. M'Nalty. This case is given in abstract at p. 736. But it must be remembered that syphilis is very common amongst civilians, those at least who frequent our hospitals, yet aneurism is not proportionately or remarkably prevalent amongst them; whilst, on the other hand, disease of the aorta seems to be a special appanage of the soldier, for which adequate causes relating to his dress and discipline may be adduced. It must be added that no distinction, based upon symptoms or morphological peculiarities of structure, has been established, or even advanced, between ordinary atheroma and so-called syphiloma of the arterial coats. I think, then, the allegation that atheroma is identical with syphi-

\* *Dublin Quarterly Journal*, August, 1871.

† *Guy's Hospital Reports*, series iii., vol. xiii., 1867.

‡ *Army Blue Book*, 1870.



loma, and that the frequency of aneurism amongst soldiers is explained by the prevalence of syphilis amongst them, is not proven.

*Myocarditis*, or inflammation of the substance of the heart, may be presented in the *acute* or in the *chronic* form. In both, the investing or the lining membrane of the heart is at the same time engaged, and occasionally, both the one and the other. The possibility of parenchymatous inflammation of the heart, unaccompanied by pericarditis or endocarditis, has been actually called in question by respectable authorities.

Neither Corvisart nor Bouillaud had met with an example of inflammation of the substance of the heart uncomplicated with peri- or endocarditis.

Stokes barely admits the possibility of its occurrence in an isolated form, but he has not met with an example, nor have I.

It will not be difficult to understand why the isolation of the muscular substance of the heart in the inflammatory process should be so rare, if indeed it be possible, when due consideration is given to the fact, that on both its surfaces it is not only closely invested by serous membrane of extreme delicacy of structure and susceptibility to irritation, but that the deeper or areolar layer of this membrane is structurally continuous with its connective tissue. Having myself not witnessed an example of myocarditis in which the pericardium or the endocardium was not likewise in some degree involved, and having seen no well authenticated case of it on record, I feel bound to regard such a contingency as outside the domain of established pathology.

The occurrence of inflammation of the substance of the heart to a limited depth, in connexion with pericarditis and endocarditis, is, on the other hand, so far from being uncommon, that in my opinion it constitutes the rule; and I believe that between these cases and inflammation of the walls of the heart in their entire depth, there is only a difference of degree. As to extent, it is rarely general, being most frequently confined to one or other ventricle, and usually to the left.

Laennec was not satisfied of the existence of a single well authenticated case of general inflammation of the heart.\* In-

\* *Traité de l'Auscultation Médiate*, vol. ii., p. 554.

flammation, he urges, generally augments the colour and the density of structures; whereas, in the alleged cases of general cardiac inflammation the substance of the heart was discoloured and softened.

Corvisart and Bouillaud, however, give several examples, and Rindfleisch gives one, of diffuse or general inflammation of the heart, in the person of a man aged fifty-four years, who was the subject of secondary syphilis. The muscular structure was heightened in colour "with a dash of violet," and increased in density; the section resembling caoutchouc in firmness, and like it exhibiting a certain iridescence. There were spots of ecchymosis under the endocardium and the pericardium.\*

Mr. Stanley published a very remarkable example of suppurative myocarditis, which, however, was associated with pericarditis, and, as I believe, consecutive to it. The subject, a boy of twelve years, and an inmate of Christ's Hospital, became feverish, and on the following day had pain in one of his knees and in his head, accompanied with delirium, and followed by dyspnoea and coma. Death took place on the fourth day of illness. There was no pain in the chest, and no symptom referable to the heart. The cerebral vessels were found congested; the abdominal organs and the lungs were healthy; the pericardium contained four or five ounces of turbid serum with flakes of coagulated lymph, and the surface of the pericardium generally was reticulated with lymph. The size of the heart was natural, and on section its structure was seen to be nearly black, the fibres being soft and compressible. Distant and numerous small collections of dark pus were found in the walls of both ventricles, those near the outer surface lifting the pericardium in a few places. The walls of the auricles were soft and dark, but did not present collections of pus. The endocardium and the valves were only vascular throughout.†

Doctor Thomas Salter furnished the particulars of a case of suppurative myocarditis confined to the left ventricle. Here, likewise, there was pericarditis, but only in the stage of vascular turgescence. A man aged fifty years had a paroxysm of pain

\* *Opus citat.*, p. 275.

† *Medico-Chirurgical Transactions*, vol. vii., 1816.

in the region of the heart and dyspnœa after a walk ; several such attacks occurred within the succeeding four weeks, but always after exertion of some kind ; there was then continuous orthopnœa, and precordial distress of the most aggravated character ; the pulse was quick and very weak, but regular, and the sounds of the heart were very feeble. Severe pain now set in over the left front of the chest, from the clavicle downwards. Death took place by asthenia. The right pleural cavity was found to contain some serum ; the left lung was attached to the walls of the chest by recent false membrane, and was in the first stage of pneumonia ; the pericardium was generally vascular, but contained neither lymph nor serum ; the endocardium and the valves were normal. The substance of the left ventricle, except to the depth of a few lines on either surface, was soft and yellow, and its entire thickness was pervaded by small abscesses, from the size of a pin's head to that of a pea. The ascending aorta was atheromatous.\*

Doctor J. Risdon Bennett quotes from the *Bulletin de l'Academ. Roy. de Médecine*, April 11th, 1843, a case of acute carditis engaging the left ventricle, and ending in abscess which burst into the pericardium. The case was published by M. Gintrac of Bordeaux. The patient was a man aged sixty-eight years, and subject to palpitation, which became very severe after a violent fit of passion, and was followed by orthopnœa and œdema of the lower limbs. The action of the heart was violent and irregular, and the sounds extensively diffused, but no murmur was audible. There were, however, loud ronchi. Pulse small and frequent, surface cold, and countenance expressive of anxiety. The pericardium was slightly reddened, and contained a turbid reddish fluid like a mixture of pus and bloody serum. The heart was large, and covered on the outer surface with a layer of concrete pus. Over the left ventricle the pericardium was opaque and easily detached, and here an aperture was found leading obliquely downwards into a large cavity corresponding to the lower portion of the left ventricle, which was separated from the upper part of the ventricle by a thick wall of unorganized lymph,

\* *Medico-Chirurgical Transactions*, vol. xxii., January, 1839.

and containing a mixture of grumous blood and pus. It is impossible to say whether the pericarditis was primary or secondary in the foregoing case, although it was assumed to have been secondary to the irruption of pus into the pericardium. He quotes from the *Gazetta Medica di Milano*, January, 1844, another example of suppurative myocarditis with pericarditis. The author was Dr. Dubini; the patient, a female, was ill only four days, and complained of feverishness, dyspnœa, and a feeling of weight at the precordium. There was a double *bruit* at the base of the heart, rough and extended in the course of the aorta; subsequently the sounds were feeble, and the pulse failed. The muscular structure of the left ventricle was yellow and infiltrated with pus; the aortic valves were inadequate, and the apex of the heart was adherent to the pericardium. The writer refers to a third case of purulent infiltration of the heart which occurred in the same hospital four days later.\*

Graves has given a case of inflammation of the heart ending in suppuration. This, he remarks, is very rare, because inflammation of the heart generally proves fatal before suppuration can take place. A man of fifty-five years, and of full habit, had been for some months suffering from cough, dyspnœa, and palpitation. Anasarca followed, with great pain and distress referred to the heart, and darting over the chest. The pulse was irregular. There was loud *bruit de soufflet* and *frémissement cataire*. The diagnosis of hypertrophy and dilatation of both ventricles was made. Death took place suddenly a few weeks subsequent to the date of the foregoing report. There was effusion into both pleuræ. The heart was enlarged, and there was general adhesion of the pericardium to the heart by means of long bands of lymph, easily broken down except at the apex, where they were strong and firm. There was an abscess in the wall of the left ventricle, large enough to contain two ounces of pus, lined by a membrane, and opening into the pericardium by a rent near the apex. Both ventricles were thick, and the valves generally unsound; those of the aorta were ossified.†

\* *British and Foreign Medico-Chirurgical Review*, vol. xx., 1845.

† *Clinical Medicine*, second edition, 1864, p. 558.



Latham has published an example of general purulent infiltration of the heart, consecutive to inflammation, and fatal in two days.\*

Laennec has credited Benevenius with having been the first to describe an abscess in the walls of the heart, and declares that he himself witnessed only a single example, which occurred in the body of a child of twelve years. The abscess, which was in the wall of the left ventricle, was large enough to contain an almond. He mentions another doubtful example, and affirms that there are no special symptoms indicative of abscess in the heart, adding that it may be wholly unattended with symptoms, as in the case given by Benevenius.†

Bonetus has recorded several examples of suppuration of the heart.‡

According to Raynaud myocarditis most frequently engages the left ventricle, and its posterior wall by preference. The external and the internal layers are, he asserts, oftener involved than the intermediate substance. He quotes Dittrich as holding that primary acute myocarditis occurs nine times out of ten in males, and most frequently in those under thirty years of age. It may arise from mechanical violence, or may complicate acute articular rheumatism, puerperal typhus, or exanthematous fever. It may likewise follow pyæmia. Mankopff witnessed it in an epidemic of cerebro-spinal meningitis.§ Da Costa asserts that there are no symptoms or signs proper to myocarditis; or, if such exist, they have not been separated from those of pericarditis or endocarditis, either of which is likewise usually present.|| And Flint declares that its presence is not determinable during life.¶

Hope is of opinion that this condition presents no peculiar sign; but he thinks "it *may* be the cause of the feeble, fluttering, irregular, intermittent action of the heart, with suffocative symptoms, when these phenomena cannot be accounted for by

\* *London Medical Gazette*, vol. iii., p. 118.

† *Opus citat.*, p. 555.

‡ *Sepulchretum*.

§ *Nouveau Dictionnaire de Médecine et de Chirurg. Pratique*, 1868.

|| *Medical Diagnosis*, third edition, 1870, p. 360.

¶ *Diseases of the Heart*, second edition, 1870, p. 555.

the presence of fluid in the pericardium, or of polypous concretions from endocarditis.”\*

Walshe thinks that great weakness and a fluttering character of the pulse *may* be due to inflammatory softening of the left ventricle ; but he inclines to attribute these effects to the implication of the cardiac nerves. He declares that no pain, and not even an uneasy sensation, exists at the precordium, unless where pericarditis is likewise present. Yet he adds that he has no experience of the affection.†

Stokes says that of myocarditis, as distinguished from pericarditis and endocarditis, we know nothing ; but he admits that the disease may exist in an isolated form. Its pathological anatomy, he adds, is but little known, because paralysis precedes disorganization of muscle, and extensive paralysis of the heart is fatal. The most frequent cause of the latter affection is a very acute myocarditis merging into the chronic form.‡

From the preceding quotations and references, it will be apparent that, independently of the implication by contiguity of the superficial muscular strata of the heart in the ordinary course of pericarditis or endocarditis, inflammation of the muscular substance to such a depth as to constitute a veritable myocarditis, though comparatively rare, is by no means absolutely so. It is further manifest, that as to the existence of symptoms and signs positively diagnostic of myocarditis, the greater number of writers hold a negative opinion.

Nevertheless, I venture to advocate the affirmative view ; I believe the diagnosis of myocarditis is quite practicable, irrespectively of the accompanying inflammation of the investing or the lining membrane, inasmuch as there are both symptoms and signs proper to it. If it can be shown that amongst the ordinary symptoms and signs of pericarditis and endocarditis, as they are witnessed every day in our hospitals, rapid failure of the pulse, suppression of the impulse and sounds of the heart, extreme precordial anguish, respiratory distress, and a sense of impending suffocation, are not to be included unless where peri-

\* *Opus citat.*, p. 197. The italics are mine.

† *Opus citat.*, p. 260.

‡ *Opus citat.*, p. 109.

cardial effusion, cardiac thrombosis, or pulmonary complication exists, I think the actual presence of such symptoms, in association with pericarditis or endocarditis, would justify the inference that the substance of the heart was implicated in the inflammatory process.

I think I may assume that the above mentioned phenomena do *not* belong to the ordinary serous phlegmasiæ of the heart, *per se*; further, that they are of not very unfrequent occurrence in connexion therewith, where none of the secondary causes just mentioned can be adduced in explanation of their presence; and, finally, that in cases fatal in an early stage, where none of these consecutive or collateral accidents have occurred, indubitable evidence of parenchymatous inflammation of the heart may be always readily detected.

The evidence upon which I rely as warranting the diagnosis of myocarditis is the following. Actual rheumatic arthritis with manifest implication of the heart, or exposure to cold quickly followed by pain in the region of the heart, and, probably, at the same time, in the limbs and in the head; delirium; virtual or absolute failure, with extreme rapidity of the pulse; pallor, coldness, and clamminess of the skin; anxious expression and collapse of the features, and tendency to deliquium; not unfrequently incomplete paralysis of one side of the body; orthopnoea and gasping respiration, with an urgent feeling of want of air, and frequent sighing; pain and great oppression at the precordium; fluttering and irregular action of the heart, with occasional intermissions; feeble or suppressed impulse; faint, foetal, and all but extinct sounds; area of precordial dulness not more than normal in extent, with or without the acoustic signs of pericarditis, of endocarditis, or of both.

The foregoing symptoms and signs are rarely all associated in the same case. Those which are essential are, pallor, coldness, and tendency to syncope; failure of the pulse; pain and oppression at the precordium; orthopnoea; gasping and suspirious respiration; feeble, fluttering, rapid, and irregular action of the heart; weak but sharp sounds, both resembling the second, or of a foetal character; with a history of recent and acute accession. The actual condition of the lungs and pleuræ, the area and degree

of precordial dulness, and the absence of chronic valvular lesion, must be duly considered, as precluding acute pulmonary congestion, effusion into the pleuræ or pericardium, and endocardial thrombosis.

The following case will serve to illustrate the foregoing remarks, although I was not afforded the opportunity of verifying the diagnosis. It is an example of the affection in an extreme and fatal form.

CASE LXVII.—*Myocarditis ; Left Hemiplegia ; Death on the sixth day of Illness.*

On Sunday morning, March 9th, 1873, in consultation with Drs. McVeagh and More Madden, I saw Mrs. P., aged about forty-two years, and a chronic invalid from uterine derangement. On the Tuesday preceding she had called at Dr. McVeagh's residence in a fainting condition. He had her removed to her home, and witnessed in the course of that day several paroxysms of aggravated cardiac asthma, accompanied with severe precordial pain. She complained of numbness in the left hand and foot, and suddenly lost the use of the left side of the body.

When I saw her, she was propped up in bed, pale, and gasping for breath. The surface was cold, and there was no pulse to be felt at the wrists. Precordial dulness was not extended, and there was no perceptible cardiac impulse. The heart was acting with slight irregularity, at the rate of 160 in the minute. The sounds were both clear, but remarkably faint. The characters of the heart's action were very similar to those of the foetus *in utero*.

Doctor McVeagh suspected endocarditis with cerebral embolism. She had been taking small doses of grey powder; the precordium had been blistered and subsequently dressed with mercurial ointment, and champagne was being freely administered. Digitalis was suggested by Dr. Madden, and approved of by me. It was accordingly given in  $\mathcal{M}$ v doses, with  $\mathcal{M}$ x of spirit of chloroform every second hour. Other treatment continued.



She died of asthenia on the following day, and no examination of the body was made.

Doctor McVeagh's diagnosis of primary endocarditis was, I have no doubt, correct. The absence of murmur, in the state of extreme debility of the heart in which the patient was examined by me, is not unusual, even in cases of chronic valvular lesion. I regard the case, then, as an example of primary acute inflammation of the endocardium, rapidly and fatally implicating the substance of the heart.

*CASE LXVIII.—Albuminuria ; Paroxysmal Dyspnœa and Mitral Systolic Murmur ; subsequent Development of Presystolic Murmur ; Hypertrophy of the Heart, and Consecutive Endo-Myocarditis ; Mitral Stenosis ; Death.*

The wife of a medical man residing in the country was brought to me by her husband, in May, 1873. She was pallid, and suffered from occasional palpitation and a feeling of uneasiness about the heart. There was sufficient evidence of hypertrophy of the heart. A bitter tonic with steel was directed to be taken, and an opiate plaster to be applied to the precordium.

On the 26th of February, 1874, I again saw this lady, in consultation with Dr. Denham and her husband. She had been confined, under Dr. Denham's care, three weeks previously, of her second child, which was living, and healthy. For some time before confinement the feet and legs had been swollen. The urine was 1·027 in sp. gr., dark brown, acid, and contained a good deal of albumen, some epithelial casts, broken epithelium, and blood corpuscles. She had, nevertheless, a favourable confinement. The œdema had in some degree subsided, and the urine assumed a more natural appearance, when she began to complain of great oppression about the heart, and occasional difficulty of breathing.

When I visited her she was pale and agitated. The feet and legs were remarkably anæmic, considerably swollen, and readily pitted on pressure; there was orthopnœa; the breathing was rapid, and the slightest effort, even that of speaking, brought on a paroxysm of dyspnœa, during which respiration became still more rapid; she gasped for breath, moaned deeply, flung her

arms about, and demanded to be lifted upright, and that air should be admitted; the chest heaved, the nares were dilated, and the face wore an expression of great fear and anxiety. This condition passed away after about two minutes; but the slightest disturbance or excitement, even that involved in applying the stethoscope to the precordium, brought it on again. The pulse was 120, and small, but regular. Precordial dulness was extended to the left. The action of the heart was tumultuous, but feeble. There was a slight "jogging" or double impulse, and the sounds of the heart were obscure, and scarcely audible. The stomach was intolerant of everything taken, and the bowels were confined. A laxative enema was administered, and digitalis, in doses of ℥xx of the tincture, was tried; but the stomach immediately rejected it.

On the following day, a musical systolic murmur was audible at the apex. The patient having had no sleep for several nights, a sedative draught, consisting of Hoffman's anodyne and Battley's liquor of opium, of each ℥xx, was prescribed as a hypnotic.

On the following day I learned that she had slept nearly the entire night in the horizontal position, and within the preceding twenty-four hours she had only one paroxysm of dyspnoea.

Up to the present date (March 4th, 1874) she has continued to take this draught at night, and with the same benefit. She is now taking quinine and spirit of nitrous ether. There is an increase in the quantity of urine passed; the œdema of the feet is less; the pulse has improved in volume, and is now only 96, and the chest may be examined without inconvenience.

There has been for the last few days only one paroxysm, which occurred on the evening of the 3rd of March, and was very severe. In the course of that evening also the patient was for some time incoherent. She can take and retain solid food, and sleep in the horizontal posture, but only on the right side. The breathing is short, and the cardiac signs remain as last described; subsequently, a well pronounced and harsh presystolic murmur became audible, and continued up to her death. This murmur could not have existed in May, 1873, and its suppression in February, 1874, is readily explained by the weakness of the patient.

I have no doubt as to the nature of the case, the most noteworthy feature in which is the great relief from the urgent symptoms of paroxysmal dyspnoea, cardiac oppression and palpitation, which was derived from the use of opium and Hoffman's anodyne ether.

She died on the 25th of May, with signs of effusion into all the serous cavities. The body was not examined.

Myocarditis may be fatal in the first stage, or that of active congestion, by paralysis of the heart and asystole; or it may lead to :

1. Abscess.
2. Purulent infiltration.
3. Ulceration.
4. Fibroid transformation.
5. Aneurism.

*Abscess* of the heart, according to Rindfleisch, consists of pus corpuscles, albuminoid molecules, oil globules, and the *débris* of disintegrated muscular fibres. It is generally surrounded by a very soft layer of reddish grey material; it may detach the endocardium, or force its way between the lamellæ of the auriculo-ventricular valves, it may burst into the pericardium and so cause rapid death, or likewise into one of the cavities, most frequently the left ventricle, and cause pyæmia or embolism in some portion of the systemic arterial circulation. It may lead to aneurism, by communicating with one of the chambers; and, finally, its contents may be inspissated and encysted, undergo caseous change, and be thus tolerated by the heart. The small and disseminated foci found in the substance and on the surface of the heart in connexion with pyæmia, puerperal fever, glanders, and other forms of blood-poisoning, consist of aggregations of *vibriones*, which ultimately penetrate the muscular fibres, and usurp the place of the sarcous elements.\*

Doctor Moxon reports a case of pyæmia in a girl aged nine years, arising from suppurative periostitis of the thigh, cranium, and hand, in which multiple abscesses were found in the walls of the ventricles of the heart and in the kidneys; one of the

\* *Opus citat.*, p. 275, *et sequent.*

former was on the point of bursting into the pericardium, which was inflamed and rough with lymph, and contained several ounces of turbid liquid. He adverts to the connexion between suppuration of the periosteum and that of the kidneys and heart, and remarks that it is so constant that he believes "when peritonitis, with pyæmic symptoms, is the cause of death, abscesses in the heart and kidneys may be expected to be present."\*

Examples of both abscess and purulent infiltration are given in the preceding pages. I need not, therefore, further dwell upon that result of acute inflammation of the heart in this place. *Ulceration* is a less common consequence. The following is a good example from Dr. Stokes.

A youth of eighteen years contracted a violent pericarditis from sleeping on the damp grass while the body was still warm after active exercise; the disease proved fatal in a little over a week. His suffering from cardiac anguish was extreme. On examination of the body, general hæmorrhagic pericarditis was revealed. The heart was covered with layers of chocolate-coloured lymph, and exhibited several patches of superficial ulceration from two to three lines in diameter, and of equal depth, engaging the false membrane and penetrating into the substance of the softened heart. The muscular structure was dark, but less so towards the inner surface. He quotes from Testa an example of ulcerative endocarditis in the body of a lady of twenty-eight years of age. After death there was found acute endocarditis of the left ventricle, with ulceration extending far into the aorta, causing rupture of the latter into the pericardium and death by hæmorrhage. The heart was fatty.†

Morgagni witnessed several examples of ulceration of the heart, both external and internal;‡ so likewise did Bonetus.§

Laennec mentions an example of ulceration in an hypertrophied heart; the ulcer was one inch long, half an inch broad, and four lines deep. Death took place by rupture of the heart. There was, during life, no evidence of this formidable lesion. ||

\* *Medical Times and Gazette*, September 28th, 1872.

† *Opus citat.*, p. 109, *et sequent.*

‡ *De Sedibus et Causis Morborum*.

§ *Sepulchretum*.

|| *Opus citat.*



Hope regards ulceration as the most common cause of rupture of the heart.

Walshe says there may be secondary ulceration of the heart, from pyæmia or phlebitis. He likewise alludes to veritable apoplexy by extravasation into the substance of the heart, as a consequence of softening.\*

Myocarditis may become *chronic* and lead to fibroid transformation, to a greater or less extent, of the walls of the heart; a condition appropriately designated "cirrhosis" of that organ by Bristowe. This is usually partial, and localized near the apex or in the papillary muscles, and may result in shortening of these, contraction of the ostia, or annular constriction of the infundibulum of the right ventricle. The latter lesion is rare in the adult, but not so in the infant. Aneurism of the heart may likewise be a consequence of fibroid transformation of its walls, from localized chronic myocarditis.

In the *treatment* of myocarditis, the urgency of the symptoms, and the serious consequences in prospect, demand the most prompt and decisive measures. I would commence by the application of a dozen leeches over the precordium, and would repeat this measure on the following day, if relief from dyspnoea and cardiac oppression were not in the interim obtained and the pulse had not improved in volume and rhythm. A succession of light, warm poultices should be applied over the leech bites to encourage the flow of blood; or, if the weight of a poultice be objected to, a layer of cotton wadding or of spongio-piline may be substituted. After the abstraction of blood, the patient should be quickly brought under the influence of mercury; and the readiest mode of effecting this object is that recommended by Dr. Law, and known as his method, which consists in the administration of very small doses of calomel at short intervals. I usually give half a grain of calomel, with two grains of sugar or a grain of James' powder, every hour, and I find that twelve such doses usually suffice to produce mild mercurial action. Alcohol, in the form of wine or spirits, in very minute and oft repeated doses, is absolutely necessary to sustain the weak and failing heart. The quantity should not exceed a teaspoonful of

\* *Opus citat.*, p. 260, *et sequent.*

whiskey or brandy, given with water, or the equivalent of wine, every half hour, lest it accumulate in the blood and irritate the inflamed structures; the endocardium and lining membrane of the aorta being, in many cases, at the same time inflamed. Opium is necessary to procure sleep. A grain of the watery extract, or ℥xx of Battley's sedative, may be given, and may be repeated in two hours if not efficacious. If pericarditis constitute a principal, or even an important element of the case, a large blister should be applied early over the region of the heart. Alkaline beverages, such as soda, Seltzer, or Carrara water, should constitute the drink. These may be given with milk or a small quantity of claret. In the stage of convalescence, quinine, strychnine, and iron should be given in combination, somewhat as follows: ℞ Sulph. quiniæ, grs. xxiv; Sulph. ferr. granul., grs. viii; Liquor. strychniæ (B. P.), ℥xl; Acid. sulphur. dil., ℥xx; Syrupi limon., ʒij; Aquæ font., q.s. ad ʒviij. St. coch. mag. ter in dies. The occurrence of a rigor, accompanied with vomiting, might be regarded as indicating the occurrence of suppuration, and the symptoms of pyæmia, supervening upon those of myocarditis, as evidence that pus had found entrance into the circulation. Very little is, however, known from actual observation, of the symptoms of this accident.

*Aneurism of the heart* was a term employed by Corvisart to designate a state of dilatation of the chambers, with enlargement of the heart. The term aneurism is not now so employed, "dilatation," with or without hypertrophy, supplying a terminology adequate to the expression of these various conditions. By "aneurism of the heart" is now understood a yielding and pouching of a limited portion of the wall of one of its chambers, by which a sac is formed, communicating with the interior of the heart, and usually, but not necessarily, projecting from its outer surface.

The causes which may give rise to aneurism of the heart, and in the order of their frequency, are the following:

1. Chronic endo-myocarditis, leading to fibroid transformation of a limited portion of the walls.
2. Ulcerative endocarditis.
3. Partial rupture of the wall on the internal surface, including the endocardium.

4. Bursting of an abscess into one of the chambers of the heart, and entrance of blood into the cavity of the abscess.
5. Discharge of an hydatid cyst into one of the chambers, and entrance of blood into the hydatid cyst.

Of 74 cases collected from various sources by Thurnam,\* the left ventricle was the seat of aneurism in no less than 58. In these 58 cases there were 62 aneurisms, inasmuch as the disease was multiple in 4.

The composition of the sac-walls in the 62 aneurisms was as follows: In 15 it was composed of muscular fibres and the pericardium; in 4, of the endocardium and pericardium only; and in 25, of all the structures of the heart; hence he concludes the greater number is, in the first instance, "true." In a few, the walls of the sac had undergone cartilaginous or calcareous transformation. An adherent pericardium, which was present in 21 of these cases, affords the principal protection against rupture of the sac externally. This accident occurred in only 1 of the cases of adherent pericardium collected by Thurnam, and in this solitary instance the fatal extravasation took place into the left pleural cavity. The contents of the sac were composed of laminated fibrin in 23 instances, and in 17, of dark clotted blood.

As to the seat of aneurism, Breschet held that it was nearly, if not entirely, confined to the apex of the left ventricle.

Thurnam declares this to be too exclusive. It was located at the apex of the left ventricle in 27 out of the 58 cases already referred to; at the base of the left ventricle, in 21 of these cases; and at an intermediate portion of the ventricle in 15. The interventricular septum was the seat of the aneurism in 3 cases.

He has collected 9 examples of aneurism of the left auricle. Of these, 8 were examples of general dilatation of the sinus, and 1 only consisted of a circumscribed projection of the wall of the sinus, as large as a nut, and containing dense laminated fibrin. All 9 cases were connected with narrowing of the mitral orifice.

In reference to this last observation I would remark, that where mitral stenosis exists the usual condition of the left auricle is that of excentric or dilated hypertrophy.

He justly regards inflammation of the structures of the heart

\* *Medico-Chirurgical Transactions*, vol. xxi, 1838.

as the most frequent cause of aneurism, by leading to fibroid transformation and pouching of the wall within a limited area. This is usually of rheumatic origin. Such is likewise the opinion of Rindfleisch. As to sex, Thurnam says that of 40 cases in which the sex is mentioned, 30 were males and 10 females; whereas, in arterial aneurism males are oftener affected than females, in the proportion of 8 to 1 according to Hodgson, and 11 to 1 according to Lisfranc. The size of the tumor varied from that of a hazel-nut to that of the ventricle.

The disease is most common between the ages of twenty and thirty years, and again, in advanced life; whereas, according to Sir A. Cooper and Bizot, arterial aneurism occurs most frequently between thirty and fifty years. Mechanical injury, violent fits of anger, forcible straining whilst the breath is retained, and such causes, may likewise give rise to the disease.

Flint gives 5 examples of aneurism of the heart from his own experience.\* Of these, 3 were in persons advanced in years, and in 2 instances the age is not stated. Three were males, 1 was a female, and the sex of the fifth is not given. The left ventricle was the seat of the disease in all; the apex in 2, and the base in 3 instances. The size of the tumor did not exceed that of an English walnut. The walls of the sac were composed of the pericardium and endocardium in 2 cases, and of the pericardium and muscular substance in 1. They were calcareous in 1, and in the fifth the composition is not mentioned. In 2, the contents of the sac were laminated. In 1, the state of the heart as to volume was normal, and in 2 it was greatly enlarged; but in both of these, valvular disease was likewise present. The symptoms and signs were in all neutral, or non-suggestive of the lesion actually present.

In regard to diagnosis, Dr. Flint expresses himself as follows: "The existence of this lesion is hardly determinable with positiveness during life."

Thurnam says: "The diagnosis of aneurism of the heart must, in the present state of our knowledge, necessarily remain very doubtful. Indeed, it is not to be expected that a diagnosis will ever be effected without the aid of information to be derived

\* *Opus citat.*, p. 117.



from acoustic and manual examination." The symptoms, as classified by him in the order of their frequency, are as follows: Dyspnœa, in 15 instances; precordial pain, 14; dropsy, 10; palpitation, 9; anxiety, 8; syncope or syncopal tendency, 3.

From the particulars of 51 cases collected by himself, Dr. Quain concludes that cardiac aneurism is almost equally distributed in the five decades from twenty to seventy years, inclusive. Of these cases, 1 occurred in a child of twelve years, and 1 in a person aged eighty-two years. He further concludes that the liability of females is to that of males as 1 : 3; a proportion identical with that given by Thurnam. As to the cause of the lesion: in 32 cases, of which he possessed the history, it arose from ulcerative endocarditis in 3 instances; from circumscribed fibroid transformation in 21; from bursting inwards of a cardiac abscess in 2; and from circumscribed fatty softening in 6.

In reference to situation, it is generally in the left ventricle; out of a total of 303 cases referred to by him, in which it was so situate, it was located at the apex in 138; at the base in 101; and intermediately between these points in 67. The duration is, he thinks, doubtful, because the diagnosis is not practicable. It is usually fatal by rupture, or by impeding the movements of the heart.\*

Dr. Cragie gives a list of 20 cases, including 1 observed by himself; in this instance the aneurism was situated at the base of the septum ventriculorum, was large enough to contain a walnut, and projected into the right ventricle, which was thereby contracted.†

Mr. Hodgson has published an example of aneurism engaging the apex of the left ventricle, nearly as large as the ventricle itself, and filled with laminated fibrin like an ordinary arterial aneurism. The sac was very thin, and contained no visible muscular fibre. The apex is, in his judgment, the usual seat of this lesion; and fibroid transformation of the wall, and tubercular deposit, its most common cause.‡

Mr. Higham Hill gives the case of a female, aged sixty-five, and thin, who was suddenly seized with acute pain in the region

\* Lumleian Lectures, *Lancet*, March, 1872.

† *Edinburgh Medical and Surgical Journal*, April, 1843.

‡ *Provincial Medical and Surgical Journal*, December 9th, 1843.

of the heart, accompanied with dyspnoea and vomiting. When seen by him, she was in a state of collapse, breathing with difficulty, and inclined to vomit. There was still pain in the region of the heart. The action of the heart was regular, but very weak. There was no murmur. She rallied under stimulant treatment, but died suddenly, twenty-four hours later, by hæmorrhage into the pericardium through a rent in the right auricle, which communicated with another rent two inches lower down on the internal surface of that chamber. The wall of the auricle between these two openings was dissected into two layers. There was general fatty degeneration of the heart, the left side being hypertrophied and the right side atrophied.\* The absence of murmur in this case can be explained only by the situation of the aneurism, the force of contraction of the right auricle being inadequate to the production of a murmur.

Staff-Surgeon McNalty recently published an example of aneurism of the left ventricle, arising, as he supposes, from syphilitic deposition in, and consequent fatty degeneration of, the heart.† The patient was a soldier, aged twenty-eight years, of very intemperate habits, and repeatedly the subject of syphilis, for which he was treated in hospital no less than twelve different times. No suspicion of cardiac disease would seem to have been entertained. This man was found insensible in bed, and died soon afterwards. An aneurism of the size of the end of the thumb projected from the apex of the left ventricle. Its walls were very thin and devoid of muscular structure, and from its cavity, which communicated with the left ventricle, and was lined by laminated fibrin, a devious passage led beneath the visceral pericardium to the base of the ventricle, and here an opening had been formed into the sac of the pericardium, through which a fatal hæmorrhage, amounting to four ounces, had taken place. The muscular structure of the heart had undergone fatty degeneration; and throughout its substance, but chiefly in the walls of the left ventricle, a yellowish-white deposit, apparently "gummatous" was presented, partly in nodules and partly diffused. Examined microscopically, this deposit was found to

\* *British Medical Journal*, April 12th, 1873.

† *Medical Times and Gazette*, June 14th, 1873.

consist mainly of ovoid cells in an obscurely fibrillated stroma. The aorta, above the valves and in the transverse portion, was atheromatous. Doctor McNalty is of opinion that the case was one of primary syphiloma of the heart, followed by fatty degeneration and aneurism of the left ventricle.

Inflammatory softening of the heart is, in my judgment, the most frequent cause of aneurism of its walls; but I cannot agree in the opinion expressed by Dr. Milner Fothergill, that the "yielding and pouching of the wall take place during diastole."\* I am satisfied it occurs during systole.

The *treatment* of aneurism of the heart should be conducted on the same principle as in dealing with aneurism of the aorta; namely, mental and physical excitement should be strictly avoided, and lamination of the contents of the aneurism should be promoted by all suitable measures. No means are more efficacious for this purpose than iron, and nutritious diet; alcoholic stimulants being strictly prohibited. Eminently unpromising though these cases be, they are not absolutely beyond hope; as at least one example of cure by calcareous transformation of the sac, and solidification of its contents, has been recorded by Dr. Wilks.

In the two following cases are presented the only examples of cardiac aneurism which have come under my notice. The first, for the advantage of witnessing which I am indebted to Doctor Stokes, was not primarily cardiac; and in the second, the diagnosis rests upon physical evidence exclusively, as, fortunately, an opportunity for testing it by *post mortem* examination has not been afforded.

#### CASE LXIX.—*Varicose Aneurism of the Heart.*

A man, aged thirty-one years, a strolling musician of very intemperate habits, was examined by me at the Meath Hospital, on the 4th, and again on the 6th of February, 1871, by the kind permission of Dr. Stokes, under whose care he was. Six years previously he had been treated by Dr. Hudson in that hospital for an affection of the heart, from which date, till seven weeks prior to his second admittance, on the 18th of January, 1871, he had enjoyed good health with the exception of slight dyspnoea.

\* *The Heart and its Diseases*, 1872.

About that time, after hard drinking and exposure to severe cold, his breathing became more oppressed and his feet swollen. He was now, by the advice of a dispensary physician, put under the full influence of mercury, and not deriving benefit therefrom, he sought admittance for the second time into the Meath Hospital.

When I first visited the man his condition was the following: The face and neck were puffed and livid; the lower limbs congested and swollen; there was urgent dyspnoea, which was accompanied by constant teasing cough. The lungs were oedematous and the liver engorged. The pulse was 108, weak, and irregular. Precordial dulness was extended, and the cardiac impulse diffused. At midsternum, a strong fremitus was perceived by the hand, and here, likewise, a double "booming" murmur was heard; the first was systolic in rhythm, and the louder of the two; the second was diastolic, and both completely replaced the normal sounds of the heart. The poor man informed me, that for some weeks previously he had been himself greatly disturbed by these sounds, which he compared to those made by "a woman churning."

On the occasion of my second visit, two days later, the fremitus was not to be felt at midsternum; but, in this situation, the double murmur was still audible, although manifestly by transmission only, and much less distinct; whilst at the apex, which pulsated feebly in the fifth interspace and nipple-line, two murmurs of identical quality and rhythm, but much louder, were heard. I was informed by Mr. Murphy,\* the efficient and accomplished clinical clerk, that previously to that day a very faint and soft blowing murmur was alone audible at the apex. No abnormal impulse was anywhere detected. Both murmurs were distinctly audible over the entire left front, but not in the left back, or anywhere on the right side of the chest. There was no dysphagia, and no difference in the respiratory sounds on the two sides. I made the presumptive diagnosis of aneurism of the primary portion of the aorta, opening into one of the ventricles of the heart.

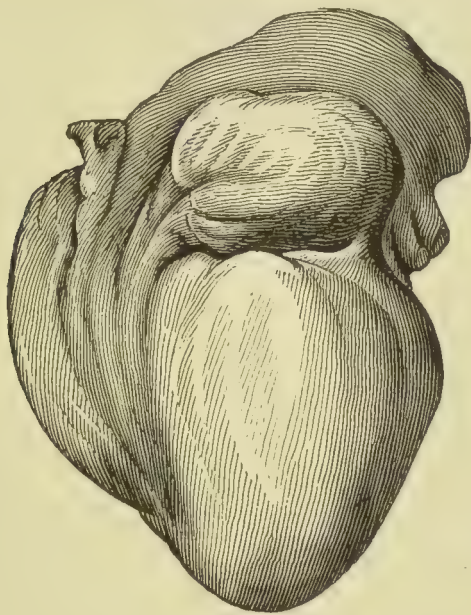
The patient died a few days subsequently, and the details of the case, with the viscera, were laid before the Pathological Society, on the 18th February, 1871, by Dr. Stokes, from whose

\* Now a distinguished officer in the Indian Medical Service.



report the following particulars are borrowed. "The aorta was greatly dilated, and its tunics much diseased. From the anterior wall of the vessel, close to the semilunar valves, there sprang an aneurism which had attained nearly the size of a tennis ball; it may be said to have arisen from one of the sinuses of Valsalva. In its posterior wall there was a rounded opening through which a goose-quill could be passed. This aperture established a communication between the aneurismal sac and the right side of the heart, for the opening conducted into the conus arteriosus of the right ventricle, immediately below one of the semilunar valves of the pulmonary artery. The sac, which did not contain coagula, lay within the pericardium." (*Vide* Figs. XLII. and XLIII.)

FIG. XLII.



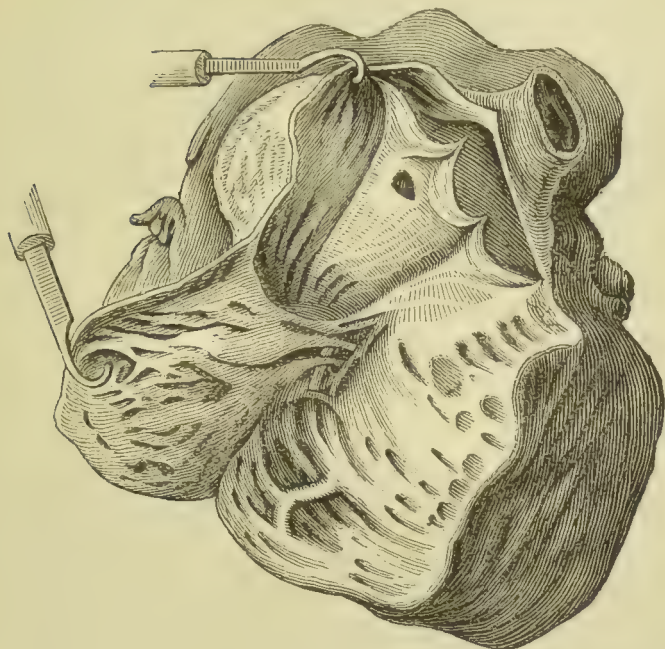
Aneurism of heart. Posterior view. (Case 69.)

At the request of Dr. Stokes, and with the permission of the President, I made the following remarks, as quoted from the *Proceedings* of the Society,\* in reference to the grounds upon which the diagnosis of aneurism of the aorta, communicating with one of the ventricles of the heart, was based. "Dr. Hayden said he had but little to add to the very clear statement which Dr. Stokes had made with regard to this extremely interesting case. If he could correctly analyze his own impressions, and it was not

\* *Proceedings*, vol. iv.

always easy to do so in retrospect, he might say that the impression of aneurism had existed in his mind from two sources. The first was that the murmur of exit was of a character which he had never heard in association with simple valvular disease. There had been a tendency of late to decry the independent value of the quality of a murmur, but he had always held that this was of the utmost importance in diagnosis. The murmur of exit at the base was of a *splashing* character, or such as would result from the sudden entrance of a body of liquid into a dense-walled and resounding cavity. Secondly, he inclined to the opinion that this murmur was not due to obstruction at the aortic orifice, mainly because it was not transmitted into the carotids. This seemed a point of some interest in differential diagnosis. He had not met with a single example of systolic murmur at the base, proved by dissection to have been due to disease of the aortic valves, in which the murmur was not distinctly transmitted into the carotids; this he regarded as a crucial test in doubtful cases. He had, however, repeatedly met with examples of basic systolic murmur not transmitted into the vessels of the neck; but in all such cases the cause of murmur resided in the aorta itself, and not in the valves. On the second day he examined this case, he found that the changes Dr. Stokes mentioned had taken place in the *interim*; namely, complete transfer of fremitus from the base to the apex of the heart. On the second day, likewise, the murmur that had existed at the base at the date of the first examination, was audible there only by transmission, and was now, according to his judgment, heard at the apex with undiminished intensity and unaltered character. This seemed to him a confirmation of his original view; for he could not, on any other assumption, comprehend the sudden and complete transfer of the thrill and murmur from the base to the apex of the heart. He could not, in short, reconcile these two facts with any other view save that of aneurism. Of course the diagnosis was rather presumptive than positive; that is to say, that the perfect confidence which one feels in dealing with familiar facts, was not experienced, owing to the rarity and eccentric character of the phenomena here presented. With the above reservation, the diagnosis was, great dilatation and advanced atheromatous transformation of the

FIG. XLIII.



Aneurism of heart. Interior of right ventricle. (Case 69.)

aorta, and aneurism of the aorta in the vicinity of the valves, establishing a communication with one of the ventricles of the heart."

I am indebted to Dr. Stokes for the use of the engravings from which the annexed impressions (Figs. XLII. and XLIII.), illustrative of the foregoing case, have been taken. Fig. XLII. represents a posterior view of the heart and aneurismal sac unopened; and Fig. XLIII. shows the interior of the right ventricle, with the aperture in the septum ventriculorum immediately below the right sigmoid valves, by which the aneurism had established a communication with the right ventricle.

By the kindness of my colleague, Dr. Nixon, under whose care the patient was admitted, I have had frequent opportunities of examining the following very interesting case. For the diagnosis I am alone responsible.

CASE LXX.—*Double Apex-Murmur ; Diastolic Apex-Thrill ; Reduplication of Second Sound. Diagnosis: Aneurism of the Left Ventricle Located at the Apex, and Mitral Regurgitation.*

James T., aged seventeen years, porter in a merchant's store,



was admitted into the Mater Misericordiæ Hospital, under Dr. Nixon, in the last week of May, 1873. He was delicate looking, with red hair, and very pale skin. Seven years previously he had an attack of rheumatic fever, in the course of which the heart was probably engaged, as he spoke of pain and distress experienced at the time in the precordial region. Six months prior to the date of admittance, he began to suffer from great weakness and shortness of breath.

When I examined him, the pulse was exceedingly weak and irregular, and not registerable at the wrist. The cardiac pulsations were 120 in the minute, irregular, unequal, and intermittent. Respiration 24. Precordial dulness somewhat extended to the left inferiorly. Apex-pulsation in the sixth inter-space, half-an-inch outside the nipple-line. The hand placed here experienced a "buzzing" thrill, synchronous with diastole, and accompanied by a movement of elevation and expansion over a space about the size of a shilling. At the point of apex-pulsation a double murmur was heard. The first was strictly systolic in time, soft, and blowing in quality. At a subsequent examination (11th June) it was of a musical character; it replaced the first sound, was loudest at the apex, and was likewise audible in the left axilla and back. The second murmur was postdiastolic, in the sense of accompanying the latter element of a double second sound; it was of a slightly booming character, and was strictly confined to the area of apex-pulsation. At the base an ill pronounced first sound was heard; also a double second sound, the latter element of which was remarkably loud and sharp in the pulmonary artery.

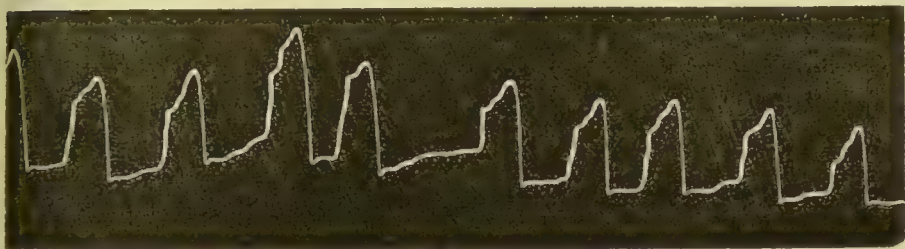
The boy is still (June 13th, 1873) under observation. The grounds upon which the diagnosis of aneurism of the left ventricle at the apex was made, are briefly the following. The diastolic murmur was of strictly apex-origin; an apex diastolic (not *postdiastolic*) murmur, is one of the rarest phenomena in cardiac acoustics, and can, in my judgment, only be due either to an abnormal communication between the aorta or pulmonary artery and one of the ventricles, as in Case 69, or to an aneurism springing from the wall of either ventricle. The former supposition was obviously excluded in this case, and the latter alone remained



to explain the phenomenon. This supposition was in my mind raised to conviction, by the existence at the apex of a diastolic impulse, of limited extent and expansile character, accompanied by a buzzing thrill, and by the elongation of the heart *without* aortic patency. That the systolic murmur was due to mitral regurgitation, I considered proven by its being audible in the axilla and left back, and by the intensification of the second sound in the pulmonary artery.

For the two following illustrations of the action of the heart and pulse in the preceding case, I am indebted to Dr. Nixon.

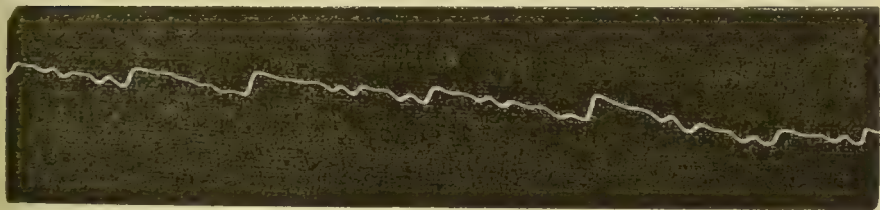
FIG. XLIV.



James T.

Cardiac tracing. Aneurism of left ventricle of heart. (Case 70.)

FIG. XLV.



James T.

Pulse tracing. Cardiac aneurism. (Case 70.)

*Wounds of the heart\** may be considered under two heads; namely, those implicating the pericardium alone, and those engaging the heart.

Wounds of the pericardium are most serious; they are scarcely less fatal than wounds of the substance of the heart. They very rarely occur without implicating the heart itself, owing to the close adaptation of the investing membrane to the con-

\* Though not belonging to the category of "Diseases of the Substance of the Heart," wounds of that organ will be most conveniently discussed in this chapter.

tained organ, equally in systole and diastole. Not only, however, *may* the pericardium be wounded without injury to the heart, but the heart may be wounded, even mortally, whilst the pericardium has sustained no apparent injury; having yielded before the impingent body, such as a bullet or other blunt missile or weapon, and been driven by it into the wound of the heart. At least four such examples are on record: one by Borellus; one by Professor Holmes of Montreal; one by Dr. Ward of Manchester, and a fourth by Dr. Fraser of the London Hospital. In the first case, which was described by Latour, a person was shot by a ball, and lived for three or four hours. The pericardium was found pushed into the wound in the heart, and, though filled with blood, was itself uninjured.\*

Sir A. Cooper relates the following case of wound of the pericardium. A man was wounded with a reaping-hook which penetrated the pericardium; he survived about three weeks. A mixture of blood and pus was found in the pericardial sac.

Hennen relates the following. A man received a bayonet thrust through the diaphragm; he survived three months, and then died of pneumonia. The pericardium was penetrated, but the heart was uninjured.†

Doctor Parkes gives a singular case of this kind. A juggler, whilst exhibiting the trick of swallowing a sword two feet long, three inches broad, and round and blunt at the extremity, finding the weapon stopped low down in the œsophagus, violently pushed it onwards, and wounded the pericardium at its reflection from the right auricle. He immediately jumped from the ground, and fainted. Thirty minutes afterwards he complained of pain in the region of the heart and a systolic frottement was heard at the apex; this was double on the following day, and audible over the entire precordium. Percussion-sound was abnormally clear, no doubt from the presence of air in the pericardium. Death took place on the third day.‡

Wounds of the heart may be conveniently divided into *pene-*

\* For the references to these cases, as well as for information of the utmost value on this subject generally, I am indebted to the able and exhaustive article of Mr. J. F. West (*St. Bartholomew's Hospital Reports*, vol. i., 1870).

† *Chelius' Surgery*, vol. i., p. 454.

‡ *Transactions of the Pathological Society of London*, vol. iii., p. 41.

*trating* and *non-penetrating*; and again, each of these classes may or may not be associated with *transfixion* of the heart.

Wounds of the heart, of whatever kind, may be *immediately* or within a few minutes, fatal from hæmorrhage. They may be *proximately* fatal, on reaction supervening, by dislodgment of a clot from the wound, or by pressure upon the heart from a coagulum formed in the pericardium, the result of a gradual oozing of blood from the wound; or *remotely*, by inflammation of the pericardium or the heart, or by the structural alterations resulting therefrom.

Fatal primary hæmorrhage usually proceeds from the chambers of the heart; but, when fatal at a later period, the bleeding is most frequently derived from a wounded vessel in the walls of the heart or chest.

Hippocrates declared that wounds of the heart were always fatal; whilst Galen held the modified opinion that penetrating wounds alone were instantly mortal, especially those of the left ventricle.

Ambrose Paré was the first who recorded an authentic case of wound of the heart; in that instance the wound was the result of a sword-thrust, and was large enough to admit the finger.

Muler recorded an example of wound of the right ventricle, the subject surviving sixteen days.

Job à Meeckren recorded the first case of traumatic pericarditis; and Morgagni was the first who showed that hæmorrhage into the pericardium was fatal by impeding the motions of the heart.\*

The right ventricle, from its situation, is the part most frequently wounded. Next in frequency the left ventricle. The auricles, owing to the protection afforded them by the sternum, are least frequently the subjects of mechanical injury; but wounds of them are always penetrating, and yield the most copious and rapidly fatal hæmorrhage, owing to the extreme thinness of their walls.

Out of 121 cases collected by Jamain, the right ventricle was

\* Raynaud, *Nouveau Diction. de Médecine, et de Chirurgie Pratique*, vol. viii., article "Cœur." Also Jamain, "Thèse de Concours pour l'Aggregation," *Sur les Plaies du Cœur*.

wounded in 43 instances ; and of those collected by Olivier, 61 in number, it was the part injured in 23 instances. Wounds of this chamber are nearly always produced by penetrating wounds of the chest ; whilst those of the left ventricle have occasionally resulted from the passage of foreign bodies from the œsophagus or the stomach.

Wounds of the pleuræ and lungs constitute the most frequent, and likewise the most formidable, complication of wounds of the heart. They are almost invariably fatal when complicating wounds of the heart of whatever kind. Penetrating wounds of the lung may be readily diagnosed by the escape of bubbles of air from the external wound, and by the occurrence of hæmoptysis. Wounds of the heart, however, do not admit of such ready or facile diagnosis. When instantly fatal, unless the superficial wound be directly over the heart, it will be impossible to affirm with confidence that death was caused by injury to that organ. When death follows at a more remote period, the difficulty of positive diagnosis will not be so great ; because time will have been allowed for the development of physical signs, and for the due estimation of the symptoms and other evidence actually present.

The symptoms and signs indicative of wound of the heart are the following. Faintness, or actual syncope ; terrified expression of face ; pallor ; quick and feeble pulse ; fluttering action, and sometimes pain in the region of the heart ; sighing respiration ; cold surface ; weak voice and great thirst ; and occasional nausea and vomiting. The sounds of the heart are indistinct, and a “whirring” murmur like that of varicose aneurism is sometimes heard when the cavities have been penetrated, as mentioned by Jobert ; but, manifestly, this phenomenon belongs exclusively to cases where a communication exists between two of the chambers, as justly remarked by Mr. West. The percussion phenomena vary between complete dulness, where the pericardium is full of blood, and tympanitic resonance, where it is distended with air, whether this be obtained directly from the exterior, or indirectly by communication with the lungs or alimentary canal. In the latter case there will be cough and hæmoptysis, or hæmatemesis and passage of blood by stool. Copious hæmorrhage from the ex-



ternal wound does not prove that the heart has been implicated, as it may proceed from wound of the internal mammary or an intercostal artery, or from a wound of the lung. Bleeding from any of these sources, but especially from the heart itself, whether penetrated or not, is often profuse and quickly fatal. Oftener still it ceases on the occurrence of syncope, only to return with reaction. The external bleeding may be trivial, even whilst a fatal hæmorrhage into the pericardium is actually in progress. The mind is usually clear to the end. After the second day the danger of death from hæmorrhage, whether by syncope or by mechanical arrest of the heart's action from clot in the pericardium, may be regarded as past. From the second to the fourth day, however, pericarditis may be apprehended, and, when it does supervene, its presence will be announced by physical signs identical with those which characterize the idiopathic form of the affection, allowance being made for the possible presence of blood or air within the sac of the pericardium. The results are likewise similar; that is to say, adhesion, serous effusion, suppuration, or ulceration, may follow traumatic inflammation of the pericardium as they do that which arises idiopathically. The two latter results are, however, more common when a wound has been the cause of inflammation; because, in such case, air has found access to the serous cavity, and the irritation arising from its presence has been aggravated by that of decomposing blood.

Penetrating wounds, if not immediately fatal, may give rise to endocarditis, and, more remotely, to disorganization of the valves; to softening and ulceration of the heart, or to aneurism of its walls. Finally, embolism of the systemic or pulmonary arteries may likewise arise from this cause.

The prognosis is always unfavourable, whether the wound be penetrating or not; a fatal result may, and generally does, follow wounds of the parietes of the heart implicating a coronary artery, the opinion of Senac to the contrary notwithstanding. Where primary hæmorrhage is not fatal, the proximate or remote consequences usually lead to early dissolution.

Chassaignac mentions a case in which the apex of the heart was torn off by a pistol-ball. There was no external hæmorrhage

or hæmoptysis; but the patient died of pericarditis on the fifth day.\*

Nevertheless, examples of complete recovery from wounds of the heart have been fully authenticated. Brugnoli, of Bologna, gives the case of a man who was stabbed with a knife in the left side of the chest, and died nineteen years and seven months subsequently. A scar was found on the anterior wall of the right ventricle, and the corresponding parts of the septum ventriculorum and mitral valve, so that the heart must have been all but transfixed.†

Mr. Callender has likewise recorded an example of recovery after a deep wound of the heart. A young man who had a needle fixed in his coat, had it driven into his chest in a scuffle. Nine days afterwards he applied at St. Bartholomew's Hospital, complaining of palpitation and pain extending from the apex of the heart to the left axilla, and along the inner side of the left arm as far as the elbow. Chloroform was administered, and an incision having been made in the fifth intercostal space, where a slight prominence was felt, a needle, nearly two inches long, was extracted. The point of the needle had manifestly been deeply fixed in the apex of the heart, for, after the "eye" had been exposed by division of the superficial structures, it described curves forwards and inwards, corresponding to the movements of the heart. Pain ceased immediately on the removal of the needle, a single drop of blood only oozed from the puncture, and perfect recovery followed at the end of four weeks.

Bullets may be encysted in the substance of the heart for many years. Bâch reports a case where a bullet was lodged in the heart for twenty years,‡ and Professor Hamilton, of New York, gives another, in which a bullet lay in the apex of the heart for thirty years.§

Latoni gives the following case. A soldier received a gun-shot wound in the chest, from the effects of which he completely recovered. Six years afterwards he died of a disease unconnected

\* *Gazette des Hôpit.* September 4th, 1858.

† *Presse Méd. Belg.*, July 20th, 1862.

‡ *American Journal of the Medical Sciences*, July, 1861.

§ *Medical Times and Gazette*, January 26th, 1867.

with his former injury. At the examination of the body, a bullet was found encysted in the wall of the right ventricle, near the apex of the heart, and resting upon the septum ventriculorum.\* Finally, in the hearts of animals of chase, and in game of various kinds, bullets have been found, which must have been lodged in the substance of the organ for a considerable period, without causing permanent impairment of the health or activity of the animal. Of this, the case of the stag mentioned by Harvey, in the heart of which a bullet was found lodged, furnishes an example.

Dupuytren insists upon the possibility of superficial wounds of the heart not giving rise to serious hæmorrhage, owing to the peculiar arrangement of the fibres by which such wounds are likely to be rendered valvular.† In contrast to such wounds, Mr. Adams‡ directs attention to those implicating the aorta or pulmonary artery, which are invariably and rapidly fatal, from the absence of any such arrangement of their fibres by which hæmorrhage from them could be arrested. In support of this statement, he quotes the case of a young woman who died from a penetrating wound of the aorta by a needle, one hour after the accident. Three or four punctures were found in the aorta, half an inch above the valves. The pericardium contained about a pint of clotted blood.§

In such cases as those above mentioned, in which foreign bodies are lodged in the substance of the heart, acute thoracic inflammation, even though apparently the most trivial, is likely to assume a very unfavourable character and prove rapidly fatal. Injury to the lung or pleura most seriously aggravates the danger of wounds of the heart.

In the *treatment* of wounds of the heart, exploratory probing should be scrupulously avoided, as likely to be mischievous, by dislodging clots, and favouring a return of hæmorrhage.

The positive indications are, firstly, to favour the formation of a clot in the wound, with the view of staying the further loss of blood; secondly, to prevent its dislodgment when formed; and,

\* *Diction de Méd. et de Chirurg. Pratiques*, tom. iv., p. 258.

† *Leçons Orales*.

‡ *Dublin Medical Press*, April 3rd, 1861.

§ *Medical Circular*, January 25th, 1860; and *Dublin Hospital Gazette*, February 1st, 1860.

thirdly, to watch for, and promptly meet, symptoms of inflammation.

With the first mentioned object in view, ice should be applied over the wound, which must be quickly closed, and not afterwards disturbed, unless great dyspnœa occur, or other evidence of copious hæmorrhage into the pericardium be presented. In that case it may be necessary cautiously to remove the clots, which impede the motions of the heart. Foreign bodies should be carefully removed, without violence, and, as far as possible, without causing further loss of blood. Complete rest should be enjoined, and restorative measures *cautiously* adopted in view of the danger of secondary hæmorrhage from reaction. Acetate of lead should be given to favour coagulation, and morphia, by the mouth or sub-cutaneously, to moderate nervous excitement. Leeches may be necessary to avert or subdue subsequent inflammation, but general blood-letting should be deprecated. With the same object in view, mercury should be given. In the after treatment of the case, it may be necessary to remove serous effusion from the pericardium by mechanical means; but, this should be done with caution, considering the danger of secondary hæmorrhage from disturbance of the clot in cases still recent, and of exciting primary or secondary inflammation.

The weapons, or implements, with which wounds of the heart have been inflicted are of the most varied character. Thus, knives of various kinds, swords, bayonets, stilettoes, cobbler's awls, sharp iron bars, reaping-hooks, needles, thorns, fish-bones, and fractured ribs, have been the agents employed, or accidentally brought into requisition.

Mr. Adams gives the particulars of a case of wound of the heart by a fractured rib. The subject of the accident was the riding mare of a medical man. The animal, whilst moving at a rapid pace, fell under its rider, a heavy man, but was able to resume the journey. Twenty-five minutes afterwards it staggered and fell, and quickly died in convulsions. A double fracture of one of the left ribs was found, and the sharp end of the detached fragment had entered the pericardium, and wounded the coronary vein, whence a fatal hæmorrhage had proceeded.\*

\* *Loco citat.*



Fischer gives a table of 452 cases,\* 87 of which he divides into three categories, according to the period of advent of syncope. Of these 87 cases, syncope was immediate in 30, and lasted from two minutes to four hours; in 38 instances, collapse was postponed for a short period; in 19 instances it was still later in appearing, and was due probably to dislodgment of the clot; it occurred in a few cases so late as the twenty-eighth day, when it was fatal.

In regard to seat of injury, Jamain, as quoted by West, gives the particulars of 121 cases.

## OF THESE, THE

Right ventricle was wounded in	...	...	43 instances.
Left ventricle	...	...	28 "
Right auricle	...	...	8 "
Left auricle	...	...	2 "
Point and base of heart	...	...	7 "
Interventricular septum	...	...	2 "
Both ventricles	...	...	9 "
Both auricles	...	...	1 "
Left auricle and ventricle destroyed	...	...	1 "
Whole heart destroyed	...	...	1 "
Coronary artery	...	...	2 "
Without precise definition of seat	...	...	5 "
With no information as to site	...	...	12 "

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 121

Of Olivier's 61 cases, the right ventricle was the seat of injury in 23, and the left ventricle in 12 instances.

The following Table, intended to show, first, the seat of injury; secondly, the nature of the wound; thirdly, the period of subsequent survival of the patient; fourthly, the immediate cause of death; and, fifthly, the proportion of recoveries, has been constructed from the statistics of Mr. West, the records of the Dublin Pathological Society, and from *The Medico-Chirurgical Transactions of London*, conjointly.

\* *Holmes' Surgery*, second edition, vol. ii.

TABLE IX.—WOUNDS OF THE HEART (TWENTY-NINE CASES).

<i>Author.</i>	<i>Site of Injury.</i>	<i>Nature of Wound.</i>	<i>Period of Survival.</i>	<i>Cause of Death.</i>	<i>Recovery.</i>
Mr. Adams ..	Left ventricle	Penetrating, with round slug from fowling-piece	Half an hour	Hæmorrhage into pericardium and pressure upon the heart	
<i>Arch. Général</i> , 1842	Through heart	Penetrat. "through and through"	19 days	Abscess of lung	
Dr. Babington ..	Sigmoid flexure of colon, stomach, liver, diaphragm, heart, and right lung				
Mr. Callender ..	Apex (left ventricle ?)	Penetrating, from sewing needle	15 hours	Hæmorrhage	Recovery in 4 weeks.
Mr. Le Gros Clark	Left ventricle	Lacerated by needle	Several hours	Hæmorrhage	Recovery.
Dupuytren ..	Heart	Non-penetrat. by frag. of sternum	1 year		
Erlangen Museum ..	Left ventricle and septum	Penetrating, from cesophagus by thorn of <i>Prunus Spinosus</i>	8 days	Hæmorrhage into pericardium and pleura	
Dr. Ferguson ..	Left coronary artery	Superficial, with cobbler's awl	10 minutes	Slow hæmorrhage	
Mr. Fleming ..	Left ventricle	Penetrating and transfixing from penknife	2½ hours	Asthenia	
Dr. J. F. Frazer ..	Right ventricle, then diaphragm	Penetrating, gunshot	26 days		
Dr. J. H. Grant ..	Right ventricle, then diaphragm and stomach	Penetrating, from revolver-bullet	8 days	Abscess of heart	
Dr. Graves ..	Left ventricle	Non-penetrating, by needle	A few minutes	Hæmorrhage into right pleura	
Mr. J. Hamilton ..	Right a. and v., left n., and base of right lung	Penetrating and transfixing, from revolver-bullet	3 months	Carditis	
Mr. Hennen ..	Right ventricle	Pin wound	A month	"A fit"	
Hosp. Sick Children	Septum ventriculorum	Needle, from right bronchus	8 days	Hæmorrhage from wound of coronary artery	
Hortius ..	Both ventricles	Non-penetrating knife wound	2 hours	Ditto	
M. Lamotte ..		Oblique non-penetrating, of both ventricles	63 hours	Pleurisy, pericarditis, pneumonia	Recovery in 7 months.
Baron Larrey ..	Both ventricles	Ditto	9 months	Anasarca and gangrene	
Dr. Leaming ..	Both ventricles, and septum	Penetrating, of needle	10 years	Shock	
Mr. Morton ..	Left base	Punctured with penknife	Some time	Hæmorrhage into pericardium and pleura, and between pleura and chest-wall	
Dr. Mublig ..	Both ventricles, and septum	Penetrating, sillecto	24 hours	Pericarditis	
Dr. Neill ..	Left ventricle	Non-penetrating, of needle	5 days	Slow hæmorrhage	
M. Nélaton ..	Right ventricle	Contused, from pistol-shot	4 days	Hæmorrhage	
Prof. R. W. Smith ..	Right ventricle	Penetrating, but not transfixing, from clasp-knife	Immediate death	Phthisis pulmonalis	
		Non-penetrating		Hæmorrhage	
Mr. Spencer Smith	Apex of right ventricle	Penetrating	4 years & 5 months		
Mr. Thompson ..	Left ventricle	From fish-bone, passing out of cesophagus, and through diaphragm and pericardium	Immediate death		
University College Hospital	Descending coronary vein	Inched, 1½ lines deep, and non-penetrating			
Mr. West ..	Anterior wall of right ventricle	Extensive lacerated, by end of broken ribs and sternum			
" ..	Both ventricles				
" ..					

The subject of *malformations of the heart* would be more appropriately discussed in a special treatise on pathology, than in a work designed mainly to present a clinical exposition of cardiac disease. However, it demands some notice, both because it is the necessary complement of any work on this subject designed to be even approximately complete, and because it represents a disturbing element in diagnosis.

Andral and Paget have classified congenital malformations of the heart under the three heads of "defective," "excessive," and "perverted" development.

Peacock, whose work\* constitutes the great repertory of our knowledge of this subject, following Breschet, adopts the division into "misplacements," "malformations," and "irregularities," both in regard to the heart and the great vessels connected with it.

Misplacements are of two kinds; viz., (a) within the thorax; and (b) outside the thorax.

Malformations he regards as essentially due to arrest of development, and he classifies them according to the period of foetal life at which the arrest occurs, into: (a) Those due to arrest of development from the fourth to the sixth week; scarcely any trace of the septum between the right and left chambers as yet existing, the aorta and pulmonary artery imperfectly evolved, or the primitive arterial trunk even still retained. (b) Those which arise from arrest of development occurring from the sixth to the twelfth week; the septa being imperfectly formed, and the aorta and pulmonary artery more or less perfectly developed. (c) Those which are due to arrest at a still later period; the septa being complete, and the origin of the vessels normal, but in which from disease, the ordinary *post partum* changes have not taken place.

"Irregularities" are of two kinds; viz., (a) those arising from premature closure of the foetal passages, and (b) from anomaly in the number or form of the valves.

*Imperfection of the pericardium* has been illustrated by several cases recorded by Bristowe, Baly, Peacock, and others. In these the pericardium was represented by a crescentic fold, placed usually behind the heart; it is the product of arrested develop-

\* *Malformations of the Human Heart*, second edition, 1866.

ment of the pericardium from the sheath of the great vessels, in the direction downwards.

A *biloculate* condition of the heart has been repeatedly met with. In some cases rudimentary septa existed, and one or two ventricular vessels were always found. When only one, it corresponded to the aorta or pulmonary artery, according to the ventricle represented by the vessel actually present; and from this single vessel systemic or pulmonic branches were given off, as the case might be.

In *triloculate* hearts there are two auricles and one ventricle, the auricular septum being imperfect, and that of the ventricles either entirely absent or rudimentary. There are, in such cases, usually two auriculo-ventricular openings, but sometimes only one. Most frequently two vessels arise from the common ventricle; but, occasionally one only is so given off, representing the aorta, and from this the pulmonic vessels are supplied with blood through the ductus arteriosus. Patients with this anomaly have lived to the age of twenty years.

In the *quadriloculate* heart, the auricular, or the ventricular septum, or both, may be imperfect. The defect is most frequently found in the "undefended space" at the base of the interventricular septum, where naturally the wall consists only of two layers of endocardium, some fibrous tissue on the left side, and a few muscular fibres on the right. In this situation the opening is usually triangular, but occasionally it is oval or round. The aorta and the pulmonary artery are distinct, but the latter is often rudimentary, the pulmonary vessels being derived from the ductus arteriosus. The foramen ovale, as well as the ductus arteriosus, may be pervious; the ventricular septum is misplaced, usually deviating to the left, so that the aorta arises in part or wholly from the right ventricle, and the latter chamber, having imposed upon it the duty of carrying on the double circulation, is greatly thickened and dilated, whilst the left ventricle is imperfectly developed or rudimentary.

Hunter was of opinion, and Peacock likewise holds, that the primitive defect consists in contraction of the pulmonary artery, in consequence of which the septum, still incomplete, is pushed to the left by the great pressure of the blood, whilst the aorta is



drawn to the right, the passage of blood preventing the completion of the septum.

Meckel, on the contrary, maintains that defect and deviation of the septum are the primary anomalies; the blood of the right ventricle, finding a ready escape through the aorta, the pulmonary artery remains undeveloped.

The obstruction may be located at the infundibulum, and consist of thickening of the endocardium and hypertrophy of the muscular wall; it may exist at the arterial ring, from contraction of the fibrous zone; at the free edges of the valves by cohesion of the segments, or in the artery beyond the valves.

An example of perforate interventricular septum, unaccompanied by any other lesion of the heart, has been published by Dr. Habershon.\* The subject was a man, aged fifty-four, of intemperate habits. He was attacked with cough, dyspnoea, and hæmoptysis, after exposure to cold; dropsy quickly followed. A systolic murmur was heard to the right of the sternum, at the ensiform cartilage, and in the left axilla and back. After the man's death, a funnel-shaped opening, a quarter of an inch in diameter, was found in the base of the septum ventriculorum. That a current passed through this opening, from the left to the right side, was concluded both from the conformation of the passage, and from certain markings exhibited by a fibrinous deposit upon the adjacent segment of the tricuspid valves. In reference to cases of this kind, Dr. Habershon remarks, that the patients may enjoy tolerably good health till an attack of bronchitis deranges the pulmonary circulation.

*Atresia of the pulmonary artery* may be due to the presence of a septum or diaphragm at the orifice, or to atrophy and closure of the vessel itself. In such cases, the ventricular septum may be incomplete; the aorta communicating with the right ventricle, and carrying on the pulmonary circulation through an open ductus arteriosus, the right ventricle being hypertrophied, and the left auricle and ventricle atrophied. These cases are the most numerous. In other instances the ventricular septum is complete, whilst the foramen ovale is open, and through it and the ductus arteriosus the pulmonary circulation is carried on;

\* *Guy's Hospital Reports*, vol. xvii., 1872.

but when, in these cases, the ductus arteriosus is impervious, the circulation is maintained through supplementary branches from the aorta or left subclavian artery supplied directly to the lungs, or through dilated bronchial arteries. In these latter cases the right ventricle is only rudimentary, and closure of the pulmonary artery took place, presumably, after completion of the septum ventriculorum.

The subjects of these malformations rarely survive even for a few days; but, in one such instance at least, life has been prolonged to the tenth month.

Partial or complete closure of the right and left auriculo-ventricular orifices, and of that of the aorta, but not in the same subject, have been repeatedly demonstrated. Where the tricuspid orifice was partially or totally obstructed, the circulation was carried on through an open foramen ovale and ductus arteriosus, the latter receiving its supply through the aorta. In cases of obliteration of the mitral opening, the passage of blood was from the left to the right auricle; the foramen ovale and ductus arteriosus being patulous, and the latter receiving its blood from the aorta by inversion of the current, as in the former case, whilst the aorta arose from the right ventricle.

Where the aortic opening was closed, the circulation had been carried on from the left to the right ventricle through an imperfect ventricular septum, and thence through a patent ductus arteriosus into the descending aorta; here the current was divided into two portions, one conducted upwards through the arch and into its branches, and the other downwards. The subjects of these malformations usually die within a few days of birth.

*Premature closure of the foramen ovale and of the ductus arteriosus* is not rare, but both lesions are seldom witnessed in the same subject.

In the former case, the right auriculo-ventricular opening, and that of the pulmonary artery, are very large, the right auricle is dilated, and the right ventricle dilated and hypertrophied; the left auricle and ventricle are hypertrophied, the cavity of the latter nearly obliterated, and the left auriculo-ventricular opening proportionately reduced in diameter. The systemic circulation, which is highly venous, is carried on mainly through the

ductus arteriosus. Infants so affected have lived only a few hours.

When the ductus arteriosus has been prematurely closed, the foramen ovale has remained open, and the ventricular septum imperfect. In such cases the right ventricle freely communicates with the aorta; both are very large, as likewise are the tricuspid orifice and the right auricle, whilst the left cavities are much reduced. A minute dribblet of blood is supplied to each lung through the corresponding lateral branch of the pulmonary artery. Persons so affected rarely live beyond a few days. In some instances life has been prolonged to several months, and in one case to sixteen years.

*Patency of the foramen ovale* may arise from: (*a*) disproportion between the opening and the valve; this may be the consequence of absolute enlargement of the opening, of imperfect development or absence of the valve, or of both combined; (*b*) from perforation of a valve otherwise competent; (*c*) from laxity of a well formed and fully proportioned valve, owing to excessive length of the cornua; and (*d*) from simple non-adhesion of the valve.

Examples of the first three kinds are rare, and are associated with other serious malformations of the heart, whilst instances of the last are sufficiently common.

Doctor Mayne has recorded an interesting case of imperfection of the inter-auricular septum, the result of congenital deficiency of the operculum at the foramen ovale. A female, aged thirty-five years, suffered from cardiac distress, accompanied by obscure cardiac symptoms. There was wandering pain in the chest, accompanied with palpitation and short dry cough, and the countenance was pale (not cyanotic). Respiration was tranquil. The pulse was regular, soft, small, and ranging from 80 to 90 in the minute. The patient was lethargic, and slight exertion quickened the pulse and the respiration. The jugular veins were not distended, nor did they pulsate, and there was no dropsy. Perspiration was constant and offensive, but other excretions were scanty; appetite indifferent. General pulmonary resonance; respiratory murmur feeble, and but slightly augmented by deep inspiration or by coughing; no râles; precordial dulness extended; cardiac rhythm normal, and no fremitus. A

loud *bruit de soufflet*, accompanying the first sound, was localized at the sternal end of the left fourth costal cartilage. The second sound was distinct and normal. The patient lived two years subsequently, and died, suddenly, whilst crossing the court yard of the hospital.

The right auricle was found to be thick, and of twice its normal capacity; an opening as large as a half-crown piece existed in the septum auricularum. The edges of this opening were smooth, and not even the rudiment of a valve existed in connexion with it. The venæ cavæ were small, the tricuspid orifice was large, and the right ventricle, which alone formed the apex of the heart, was dilated and thickened. The pulmonary artery and its orifice were very large. The left auricle was of the usual size, but the pulmonary veins were large. The left ventricle was small, and its walls were thin. The mitral orifice was contracted, and the mitral valve thickened. The aortic orifice was small, and the valves healthy. The ductus arteriosus was pervious.\* In this case contraction of the mitral orifice was evidently the primary malformation, and the blood must have circulated from the left to the right side of the heart, through the open foramen ovale, whilst a very minute quantity only passed through the left ventricle.†

In reference to the foregoing most interesting case, Dr. Mayne remarks, that the feeble character of the respiratory murmur was most probably to be attributed to the arterial quality of the blood circulating through the lungs. In this opinion I quite concur. From the rhythm and seat of the murmur heard, and the small quantity of blood that must have passed through the mitral orifice at each systole of the left auricle, I conclude that the murmur was due to the passage of a current from the left into the right auricle, through the open foramen ovale. In that case, however, I would have expected that its rhythm should have been presystolic.

Peacock holds that closure of the foramen ovale is due partly to contraction of the opening, and partly to shortening and approximation of the cornua, by the oblique and horizontal bands

\* *Dublin Journal of Medical Science*, vol. i., 1841, and *Proceedings of the Dublin Pathological Society*, vol. ii., p. 35.

† See J. F. Meckel, *Tabulæ Anatomico-Pathologicæ*, fig. iii., plate i.



of muscular fibre described by Senac as intersecting the cornua, and running across the valve parallel to its free margin. The contraction of these fibres results partly from distension of the left auricle after birth, and in some degree also from stimulation by contact of arterial blood.

*Persistence of the ductus arteriosus* may result from: (a) obstruction in the lungs at the time of birth; the foramen ovale will, in this case, be likewise open; (b) obstruction at the left auriculo-ventricular opening; the foramen ovale is also open in this case,\* and (c) obstruction at the orifice of the aorta, or in the descending portion of that vessel, the ventricular septum being also defective.

Excess or deficiency in the number of the semilunar valves may arise from congenital malformation, or from subsequent disease. The former is less frequent, and, when present, it is usually found at the pulmonic orifice. Of 41 such cases reported by Peacock to the Pathological Society of London in 1851, 9 only were examples of excess in the number of segments, and 8 of these were located at the orifice of the pulmonary artery.

*Excess in the number* of segments seldom gives rise to obstruction, and, scarcely ever, to incompetency of the valves. Excess is, therefore, less often associated with serious organic disease of the heart than is defect in the number of segments, the latter being very seldom met with unaccompanied by the general results of valvular obstruction or incompetency.

The valves, both auriculo-ventricular and arterial, may be represented by a single curtain with a round or triangular aperture; or by two segments, one of which is duplicate by fusion. Disease may, however, give rise to corresponding changes.

*Atrophy* of the valves, by defect of the fibrous layer, or of the serous investment at the free edges, may also be met with.

*Irregularity in the origin, course, and distribution of the aorta and pulmonary artery* is rare. The vessels have been found transposed, the aorta arising from the right, and the pulmonary artery from the left ventricle. This condition is usually associated with patency of the foramen ovale and ductus arteriosus, and with an imperfect state of the septum ventriculorum. It may or may

\* *Vide* Dr. Mayne's case, *antea*.

not be accompanied by complete transposition of the viscera; more frequently it is not so accompanied.

The most common anomaly of these vessels consists in partial or complete closure of the aorta at the point of junction of the ductus arteriosus; the lower portion of the body being supplied with blood, wholly or in part, through the latter vessel.

*Defect or redundancy of the venæ cavæ, and of the coronary vessels, and irregularities in their course and connexions, are rare.*

*Cyanosis, cyanopathia, morbus cœruleus, or the blue disease, has been attributed by pathologists to two different, though frequently associated, causes; namely, general venous congestion, and intermixture of the venous with the arterial blood.*

The former doctrine was propounded by Morgagni, and supported by Corvisart, Louis, Laennec, Cruveillier, Valleix, Hasse, Rokitansky, Joy, and Moreton Stillé. The latter, by Dr. Hunter, Meckel, Gintrac, Paget, Williams, Hope, J. Crampton, and Walshe; also, with some modification, by Bouillaud and Forget. Peacock adopts the former view, which is also mine.

Peacock attributes the greater degree in which cyanosis is manifested in cases of malformation, to its being in such cases congenital; the integument being thinner, and the capillaries more dilatable in the infant than at a later period of life.

Cyanosis is most frequently congenital, or exhibited in the first years of life. In the latter case it is either developed gradually, or manifested quite suddenly. In mature age, however, it may be primary; it is then the result of shock, or of a severe blow upon the chest. According to Stillé\* it has been met with in association with the following lesions and anomalies.

1. Dilatation and hypertrophy of the right chambers of the heart.
2. Contracted state of the left chambers.
3. Heart with only one cavity.
4. Heart with two cavities, an auricle and a ventricle.
5. Heart with three cavities, two auricles, and one ventricle.
6. Persistence of the foramen ovale, or a cribriform auricular septum.
7. Perforation or absence of the ventricular septum.

\* *American Journal of the Medical Sciences*, new series, vol. viii., 1844.

8. Dilatation of the aorta.
9. Obstruction at the orifice of the pulmonary artery from contraction, or from adhesion or deficiency of the valves ; from cartilaginous or other growths at the orifice ; or from closure by membranous septa.
10. Persistence of the ductus arteriosus.
11. Contraction of right auriculo-ventricular opening.
12. Transposition of the aorta and pulmonary artery.
13. Origin of these vessels from one ventricle.
14. Origin of these vessels by a common trunk.
15. Aorta supplying heart and upper limbs, and pulmonary artery supplying remainder of body.
16. Aorta giving off the pulmonary branches.

He has examined upwards of 80 cases, and adverts to 5 out of this number, in which cyanosis existed, without any lesion or defect which would admit of intermixture of the venous and arterial blood. He arrives at the following general conclusions.

1. There is no proportion between cyanosis and the degree in which the blood is mixed.
2. Complete admixture of the blood may take place without cyanosis.
3. Cyanosis depends essentially upon any cause which, acting at the centre of the circulation, is competent to produce capillary venous congestion.

The most frequent cause is obstruction of the pulmonary artery, which, in some form, existed in all the cases collected by him, except 9 ; in these, an equivalent condition was found in the existence of other organic lesions.

Out of a total of 40 cases of congenital cyanosis, the pulmonary artery was partially obstructed or impervious in 28.

In reference to the duration of life in the congenital cases, Stillé remarks that death took place before the age of twenty years in 27 instances out of 40.

I am decidedly of opinion that cyanosis depends essentially upon capillary venous congestion, to whatever cause this may be due ; and in holding this opinion I am happy to find myself in accord with two such eminent writers as Drs. Stillé and Peacock.

It may be due to causes apparently the most opposite, but all

leading directly or indirectly to systemic venous congestion. I have known it to be caused by patency of the foramen ovale; serous effusion into the pericardium and both pleuræ; enlargement of the thyroid gland, both benign and malignant; and aneurism of the ascending aorta opening into the descending cava.

The case just alluded to (see "Aneurism," case of Michael W.), presented the most remarkable example of cyanosis which I have witnessed. The upper part of the body of the patient, a young man, was literally black, as if it had been thickly smeared over with dilute ink.

Even in cases of congenital malformation of the heart, leading inevitably to admixture of the venous with the arterial blood, it will be found, that although the ordinary condition of the body is one representing an imperfect circulation in respect to temperature, ability to bear cold, tint of cutaneous and mucous surface, and activity of secretion and of mental faculties, yet that cyanosis, or positive blue discoloration of the surface, is presented only when the mind or body is under excitement, *i. e.*, when general venous congestion is produced by quickened circulation.

Irrespectively of cyanosis, which is a symptom of much too general import to possess any special diagnostic value, congenital malformation of the heart, involving direct communication between its opposite sides, is characterized by certain general features which can scarcely be misunderstood. These are indicative of a circulation, not only feeble and imperfect, but liable to paroxysmal derangement of an aggravated character. Thus, the temperature is below the normal standard, secretion is sluggishly performed, the mental faculties are dull, the surface is pallid or dusky, and the temper is either singularly passive, or morbidly irritable. Under excitement of any kind which is capable of quickening the circulation, the surface of the body, and especially that of the face and neck, becomes swollen and livid, the breathing rapid and laboured, and the action of the heart tumultuous; convulsive seizures, not unfrequently fatal, are of ordinary occurrence, under such circumstances.

Sir Dominic Corrigan exhibited before the Dublin Pathological Society\* the heart of a girl, aged eleven years, who had been cyanosed at intervals from her birth. The foramen ovale was

\* January 9th, 1858.



patent. During the patient's last illness it was remarked that cyanosis appeared only when she sat up; in the recumbent posture the colour of the surface was natural. This, he remarks, together with the absence of capillary congestion, distinguishes true cyanosis from that due to simple congestion.

A murmur may, under these circumstances, be likewise developed, the rhythm and site of which will depend upon the lesion actually present. I do not think a murmur is necessarily associated with patency of the foramen ovale. I have not met with a single example of it which was clearly traceable to this anomaly as its cause. On the contrary, the three subjoined cases, in each of which, as judged by the symptoms and the *post mortem* appearances, a direct and free passage of blood from one auricle to the other must have taken place whilst the patient was under observation, afford negative evidence of the strongest kind.

Doctor J. W. Ogle furnishes some valuable evidence on this point.\* He gives a list of 62 hearts examined with reference to the state of the foramen ovale. In 13 of these the passage was imperfectly closed; it varied in size from a fissure or chink due to non-adhesion of the valve, to an oval or round aperture admitting the tip of the little finger. In none of these cases was there evidence of ulceration, or appearance of rupture of the valve, to account for the opening. The ages of the patients varied from sixteen to sixty years, the greater number being between thirty and forty years. Murmur was not noted in connexion with any of these cases, except one; in that solitary instance the murmur was diastolic, loudest at the root of the aorta, and manifestly due to inadequacy of the aortic valves, which was proved by *post mortem* examination to have existed. In 6 of these 13 cases, in all of which death was caused by various diseases unconnected in several instances with the organs of circulation, the heart had been carefully examined during life, and it is positively averred that no murmur whatever existed.

A seventh example of similar import is quoted from Dr. Ogier Ward. It was that of an infant which died from symptoms of suffocation eleven days after birth, having been previously cya-

\* *British Medical Journal*, June 13th, 1857.

notice; on this account the heart and lungs were carefully auscultated, but no murmur was detected. A congenital communication between the trachea and œsophagus was found after death. The pulmonary artery was dilated, and the ductus arteriosus and foramen ovale were patent.

As regards the *treatment* of cases of malformation, little needs to be said. It may be briefly summed up in the general rule, to avoid all causes of excitement, maintain a moderate temperature, promote the secretions, administer nutritious and digestible food, and, where congestion is extreme and threatens life by its excess, to draw blood in moderate quantity, by means of one or two leeches. Due precaution should be observed against dangerous or fatal hæmorrhage, owing to the debility of the patient, and the occasional difficulty of arresting the bleeding.

Subjoined are the details of three examples of this condition.

CASE LXXI.—*Cyanosis; Partial Suppression of Arterial Pulse in the Right Side of Head and Neck, and Right Upper Extremity; General Dropsy; Death. Fatty Degeneration of the Heart; Concentric Hypertrophy of the Left Ventricle; Patency of the Foramen Ovale.*

Kate B., aged twenty-five years, was received into hospital, February 24th, 1865. For some years previously her breathing had been short, and six weeks prior to admittance she caught cold and had cough with expectoration; after a few days her breathing was greatly oppressed, and her feet began to swell. For three months she had not menstruated. Decubitus was exclusively sinister. The face was bloated, and of a purple colour, feet and legs swollen, right hand and forearm also œdematous, ascites, suffocative cough with mucous expectoration. Expiration prolonged. Pulse 132. The impulse of the heart was rather vigorous, but regular, and its sounds were normal. The supra-clavicular fossæ were obliterated on both sides, and on the right side especially the jugular veins were greatly distended, but without pulsation; the apex of the lung was very prominent above the clavicle, and the superficial veins of the shoulder and chest were distended on the same side. On the left side of the

neck there was venous pulsation. The chest was universally resonant, and crepitant râles were everywhere audible. A blister was applied to the chest, and small doses of ammonia and spirit of chloroform were given.

On the 26th, the observation was made that carotid pulsation could not be felt on the right side, whilst it was distinctly perceptible on the left. Œdema had disappeared from the right hand, but continued in the forearm. The bases of both lungs were crepitant, but not dull on percussion. Over the right clavicle and sub-clavicular space there was a shade of dulness. There was no difference between the right and left radial pulse, or in the pupils, and no impairment of voice.

March 6th. Pulse in left arm 120 and weak; scarcely to be felt on the right. Face greatly congested, especially on the right side, and carotid pulsation scarcely detectable by the finger or the ear on that side, whilst it was strong and visible on the left. Over the left apex of the heart there was a faint systolic murmur, but this was not elsewhere audible. Patient generally weaker, cough more troublesome. Urine free from albumen. The following mixture was now given:  $\mathcal{R}$  Sulph. quiniæ, grs. xij; Sulph. morphiæ, gr. j; Acid. sulphur. dil.,  $\mathcal{M}$ x; Aquæ font. q.s. ad  $\mathfrak{z}$ vj. A tablespoonful to be taken every third hour, together with four ounces of wine within the same period. It was manifest that pressure was being made upon the arteria and right vena innominata by some solid body.

7th. Arteries on left side of neck bounded with great violence, but no pulsation existed on the right side. Death took place in the course of that day.

The body was examined ten hours after death. The thoracic viscera were alone inspected. There were some old adhesions of the right lung, and in the apex of the left, which was puckered on the surface, a small mass of cretified tubercle was found. There were about two pints of serum in the left pleural cavity, and about half that quantity in the right. The bronchial membrane was congested. A mass of enlarged lymphatic glands occupied the upper portion of the anterior mediastinum, and was found to press upon the arteria and right vena innominata, and upon the descending cava. This circumstance afforded the only rational explanation

of the partial congestion and œdema of the right side, and of the partial suppression of pulse in the right carotid and subclavian arteries. The heart was somewhat enlarged in its transverse diameter, and its apex was blunted by excessive growth of the left ventricle. The right cavities were normal as to capacity and thickness of walls. The left ventricle was greatly hypertrophied, its wall, a little below the base, being one inch thick, and its cavity contracted. The substance of the heart was pale in section, and some muscular fibres from the left ventricle, examined microscopically, were found in the medium stage of fatty degeneration. The fibres retained their normal outline, but were composed almost entirely of fat molecules. In a few situations, transverse striæ were faintly visible. The valves were all healthy and competent. The interior of the aorta, throughout the arch, presented white streaks, as in the early stage of atheromatous change. By an oversight on the part of my resident pupil, the roots of the great cervical vessels, with the adjacent glands, were not preserved for examination. The foramen ovale was widely patent; the valve existed, but it was shrunken, and incompetent to close the opening.

On the day preceding death, and on that day only, a faint murmur of systolic rhythm was heard over the left apex. No murmur was elsewhere, or at any other time heard, although the patient had been daily and carefully examined. This murmur was certainly mitral in origin, and, according to my view as previously explained (p. 219), dependent upon mitral inadequacy produced by yielding of the walls of a softened left ventricle at the acme of systole.

The case may, therefore, be classed with those of Dr. Ogle, as evidence against the doctrine of systolic murmur from patency of the foramen ovale, even when this is accompanied by actual passage of blood directly from auricle to auricle. Direct transit of blood must have taken place in this case, at least during the few last weeks of life. Of similar import is the following case:



CASE LXXII.—*Hypercæmia of the Face and Neck; Orthopnœa and Hæmoptysis; General Dropsy; Albuminuria; Hyaline and Fatty Renal Tube Casts; Weak Action and Obscure Sounds of the Heart, but no Murmur; Death. Hypertrophy with Dilatation, and Fatty Degeneration of the Left Side of the Heart; Widely Patent Foramen Ovale; no Obstruction on the Right Side, and no Valvular Disease; Congestion of the Liver and Kidneys.*

Mr. L., aged forty-nine years, of strong frame, and apparently of robust constitution, was admitted into hospital as a private patient, paying a stipulated sum for his support and attendance, on the 14th September, 1865.\* He had lived freely, but had not drunk to excess; he had not had gout or rheumatism, neither of which was hereditary in his family. For several years previously, he had experienced shortness of breath after active exercise, and about six months prior to admission he complained, for the first time, of palpitation. The breathing now became much oppressed, the lower extremities swollen, and he spat up a little florid blood on two or three occasions. He passed some weeks at Harrogate, where, according to his own account, a physician whom he consulted informed him that he was the subject of functional derangement, but not of organic disease of the heart.

When he first came under my notice, the following was his condition. Much nervous excitement; face rather florid, but not puffed; tongue clean; bowels constipated and distended with flatus; and some liquid in the sac of the peritoneum. Considerable œdema of the trunk, scrotum, and lower limbs. Urine passed in moderate quantity, and loaded with lithates; its sp. gr. was 1.020, and it contained some albumen. Pulse 120, not strong, but regular, and equal at both wrists. Orthopnœa, but no cough. Precordial dulness extended, especially upwards. No cardiac impulse to be felt; sounds rather dull and masked, but no murmur to be heard. The base of the right lung was dull, and

\* The arrangement under which persons in affluence were received into the Mater Misericordiæ Hospital as "private patients" was found to be attended with great inconvenience, and was much abused by those for whose benefit it was intended; I rejoice to add, it has been long since abolished.

here respiration was associated with crepitus, whilst elsewhere percussion and respiratory sounds were normal, the latter somewhat exaggerated on the left side. There was marked bulging of the right supra-clavicular fossa, to which the patient referred as the seat of occasional pain and "beating," the former extending down the right side and arm. The swelling was, however, resonant on percussion, yielded loud respiratory sounds, and was manifestly caused by ascent into the neck of the apex of the corresponding lung. Pupils slightly contracted and equal. The case was deemed very obscure, and no positive diagnosis was made. A diuretic and aperient pill, consisting of blue pill, squill, and digitalis, was given thrice daily. The lower and back part of the right side of the chest was dry cupped, the use of liquids was restricted, and perfect rest was enjoined.

On the 18th he was much excited, and I discovered that he had secreted whiskey in his lock up, and taken about a pint of it in the course of the preceding night. Had spat up some florid blood. Prepuce punctured, and discharged a large quantity of serum. To have half an ounce of the following every third hour. *R* Tinct. colchici, ℥cxx; Potass. acetat., grs. cxx; Infusi calumbæ, q.s. ad ℥viij. *M*.

19th. Medicine not taken; fully a pint of sherry consumed since yesterday, despite the remonstrance of his attendants. Colchicum was now prescribed in form of pill, in combination with blue pill and compound squill pill.

20th. Had passed a sleepless night, owing to pain and spasm in lower part of right side, extending to the liver. Urine passed in moderate quantity; it was amber-coloured, acid, sp. gr. 1.020, and contained a good deal of mucus; it exhibited under the microscope octohedral crystals of oxalate of lime, a few epithelial cells studded with fat granules, some hyaline tube casts set sparsely with oil dots, and one of small size with perfect epithelium. Is much quieter, and feels better. Fæces devoid of biliary pigment.

25th. Spasm in region of heart last night. Urine increased in quantity to three pints, alkaline, and of sp. gr. 1.015. To have ox gall, blue pill, and co. squill pill in combination; afterwards a Seidlitz powder, and to be dry cupped over the heart.

Within the succeeding few days copious effusion of serum into the pericardium took place, causing great increase in the area of precordial dulness, which extended, on the 2nd October, up to the second intercostal space. Under the operation of a blister, and still more in consequence of an abundant discharge of serum through several small openings, formed by sloughing of the integument of the legs, this was much reduced on the 4th, and at that date likewise œdema of the genitals had entirely disappeared. The urine was of sp. gr. 1.020; it was usually acid, but occasionally neutral, and once, at least, decidedly alkaline; sometimes clear, frequently loaded with lithates, and in quantity about two pints in the twenty-four hours. Bowels generally constipated, and dejecta light in colour. During the last few days of his life respiration was greatly embarrassed, whilst the supraclavicular prominence, previously noticed on the right side had subsided. Sounds of the heart rather obscure.

Death took place on the 5th October, consciousness having been retained to the end.

The body was examined ten hours after death. *Abdomen.* Slight congestion of kidneys and of liver, the latter organ being somewhat enlarged. The kidneys, I regret to say, were not microscopically examined. *Thorax.* Right lung engorged, especially at the base, and dispersed throughout it were several nodules of extravasated blood. The heart was in excess of the average size; some fat on the surface. The foramen ovale was patent, admitting the point of the index finger. The left auricle and ventricle were hypertrophied. The right and left auriculo-ventricular orifices were much dilated. The pulmonary artery was somewhat in excess of the usual diameter, but of normal size relatively to the ventricle. The aorta was of average size. Ductus arteriosus not examined. The valves were all healthy. The muscular structure of the left ventricle exhibited under the microscope complete fatty degeneration of the fibres, the outlines being distinct, whilst of the greater number the centres were uniformly clear and structureless, presenting a few transparent oil dots. In many fibres the latter were disposed in linear order, some within, and others external to the sarcolemma. No striæ whatever were visible.

There must have been a free passage of blood through the open foramen ovale, as there was no valve ; the current had most probably set in from the left to the right auricle, since the tint of the face was decidedly arterial, and the left auricle was hypertrophied. There was primary disease of the kidneys, and hypertrophy, with consecutive fatty degeneration and dilatation, of the left side of the heart.

The following case affords a further example of the same kind ; viz., of passage of blood through the foramen ovale, without murmur.

CASE LXXIII.—*Rapid Pulse and Embarrassed Respiration ; Tumultuous Action of Heart ; Crepitant Râles on Right Side ; Death. Tuberculosis of both Lungs, and Right Tubercular Pneumonia ; Patency of Foramen Ovale from Non-Adhesion of Valve ; Hypertrophy of Left Ventricle ; Valves all Healthy.*

Eliza R., aged eight years, an inmate of St. Clare's Orphanage, Harold's Cross, was admitted into hospital under my care, May 9th, 1866. Had been delicate from birth, and a fortnight previous to admittance, was attacked with her present illness. She moaned constantly, the skin was hot and dry, and the lips were livid. Pulse 158. Respiration 60. The action of the heart was tumultuous ; there was much tenderness in the precordial region, but no attrition-sound or cardiac murmur existed. Over the right back generally there was crepitant râle, but no percussion-dulness, and very slight cough. The right side was dry cupped posteriorly, and then poulticed with linseed meal and mustard, and small doses of grey powder and antimonial powder were given every third hour.

Owing to the advent of diarrhœa on the 10th, mercurial inunction was substituted for the preceding. The symptoms were then much ameliorated.

On the 12th, however, respiration was again greatly embarrassed. The face was remarkably congested, and of a leaden hue. Pulse 168. Right back still crepitant, but clear on percussion. A blister was applied here, and other treatment was continued.



The diagnosis of central pneumonia on the right side, and *probable* patency of the foramen ovale was now made; the condition of the patient was most unpromising.

She died on the afternoon of that day; and twenty-four hours subsequently the body was examined. The lungs were of large volume, gorged with blood, and studded throughout with minute tubercle, which, in the right lung, had become aggregated in a few places, and was in process of softening. The superior and the central portions of the right lung had become solid; they sank in water, but not completely, as they floated in contact with the bottom of the vessel. The remainder of the right, and the entire of the left lung, though engorged, tuberculosed, and retaining the volume of full inspiration, were resonant on percussion, and floated in water. There was some liquid in the pericardium. A large mass of decolorized fibrin occupied the right auricle, and extended into the right ventricle. The foramen ovale was patent. The spoon-shaped extremity of a large director readily passed through it from the right to the left auricle. The passage, which was covered by a valve, well-formed but non-adherent at its upper margin, led obliquely from below and from the right side upwards and to the left. The left ventricle was somewhat hypertrophied. The valves, both arterial and auriculo-ventricular, were all in a healthy condition. The spleen was rough upon the convex surface, and partially adherent to the diaphragm, on which, to a corresponding extent, minute, pale, granular deposits of lymph were observable. The liver was large but healthy, as were likewise the kidneys.

This case was an example of patency of the foramen ovale, entailing upon the child constitutional delicacy, but not giving rise to serious consequences, save, indirectly, by favouring tuberculization of the lungs, till the occurrence of acute tubercular pneumonia. The resistance then offered to the pulmonic circulation, by causing increased blood pressure in the right chambers, determined displacement of the operculum, and so caused passage of blood directly from the right to the left auricle. Cyanosis then, for the first time, appeared. The pulmonary complication was the immediate cause of death.

## CHAPTER VII.

### DISEASE OF THE LINING MEMBRANE OF THE HEART.

UNDER this head we will consider: (*a*) Endocarditis, and (*b*) Structural Disease of the Valves.

*Endocarditis* may be either acute or chronic. In the acute form it is met with almost exclusively in connexion with articular rheumatism, of which it constitutes the most formidable complication. It has occurred in 16 per cent. of Dr. Peacock's cases, or in the proportion of 1 : 5·9.\* Bamberger estimates the proportion at 1 : 20.

It occasionally, though very rarely, constitutes the *only* manifestation of the rheumatic diathesis. I have met with a few such examples in persons decidedly rheumatic, as evinced by previous attacks of the disease in the ordinary form. It has been assumed also that acute endocarditis may occur in the simple idiopathic form, that is, unassociated with other antecedent or concurrent cardiac lesion, and not attributable to any of the usual constitutional causes. I have never met with an example which could be fairly adduced in support of this doctrine, and I question its truth.

It is not to be doubted that, in certain cases, pericarditis may arise from exposure of the body to cold, when over heated. Of this, Dr. Stokes has given a notable example, already referred to (p. 752). It occurred in the person of a young gentleman who, when much heated from athletic exercise, imprudently lay down and slept upon the ground without having dressed. But of endocarditis from this or any similar cause, there is not, as far as I know, an authenticated example on record.

In rheumatism, the liability to endocarditis is in direct proportion to the severity of the articular affection, and the number of joints engaged; *i. e.*, it is proportionate, presumably, to the

\* *Medical Times and Gazette*, August 2nd, 1873.

quantity of the special morbid matter actually present in the blood. Next, in point of frequency, it occurs, although not so often as pericarditis, in connexion with Bright's disease, and in this association it is usually fatal. It occurs, likewise, very frequently in measles, and less often in scarlatina, small pox, puerperal fever, phlebitis, and pyæmia. In the two last mentioned connexions, the right side of the heart alone is usually engaged.

M. Lancereaux has described a special form of ulcerative endocarditis, which is produced, as he believes, by paludal miasm.\* It usually engages the aortic valves in the first instance, but may extend to the mitral, and by preference to the anterior segment. The valves become swollen, and are soon studded with vegetations, consisting of perishable embryonic tissue, which quickly undergoes fatty metamorphosis. The valves are then softened, and may be torn by the pressure of the blood; they may undergo ulceration, or become the seat of aneurism. Abundant deposit of fibrin takes place upon the damaged valve; this may be subsequently wafted by the blood current into a remote vascular district, constituting arterial or capillary embolism, or it may undergo fatty change and disintegration, the molecular detritus so produced becoming a further source of blood poisoning. In the *débris* of these deposits M. Lancereaux has found parasitic forms resembling vibrios. The symptoms are those of septicæmia, and the disease runs a rapid course. On theoretical grounds, he suggests the use of phenic acid as the most appropriate treatment.

I have not met with a case of endocarditis traceable to the cause mentioned by Lancereaux. Examples of the disease of septic or pyæmic origin, are not, however, of unfrequent occurrence; but, as already stated, the inflammation is, in such cases, usually located on the right side of the heart.

With regard to the relative frequency of the several forms of acute inflammation of the heart, Dr. Stokes thinks endo-pericarditis the most frequent; in the isolated form, however, he considers that pericarditis is of more frequent occurrence than endocarditis. Flint and Niemeyer hold the opposite opinion. No doubt if all, or even the majority of, the examples of valvular dis-

\* *Archives Générales de Médecine*, Juin, 1873.

ease were admitted as evidence of antecedent *acute* endocarditis, the preponderance of this, in an isolated form, would be unquestionable. But at least one-half the cases of valvular lesion met with in practice are traceable to *chronic* inflammation, arising from strain, abuse of alcohol, or the rheumatic or gouty diathesis. It should be likewise borne in mind, that whilst secondary endocarditis, or chronic valvular disease with temporary excitement of the heart, may be readily mistaken for acute primary endocarditis, no such error is likely to be committed in regard to pericarditis, which, in most instances, involves adhesion of the pericardium in the first attack. Hence, a liability to over estimate the actual numerical proportion of acute primary endocarditis; whilst, in regard to pericarditis, the opposite error may be readily committed.

A further element of derangement is furnished by the occurrence, by no means infrequent, of a soft systolic murmur at the apex in the advanced stages of atonic rheumatism, or in the early period of convalescence, *not* from endocarditis, but from simple valvular inadequacy of a non-inflammatory character, and resulting, according to very high authority,\* from ataxy of the papillary muscles. I believe, however, that the murmur in question arises from atony and partial yielding of the wall of the left ventricle at the acme of systole. Excluding all such cases as those just referred to, and admitting for comparison only examples of simple acute primary endocarditis on the one hand, and of simple acute primary pericarditis upon the other, I have no doubt the latter preponderate numerically.

Acute endocarditis is confined in extra-uterine life to the left side of the heart, cases of traumatic and septic origin being excepted; in the foetus it is quite as strictly limited to the right side. The right chambers of the heart were primarily and exclusively affected in only 1 per cent. of the cases brought under the notice of the Pathological Society of Berlin, within the three years, 1868-71, inclusive. The tricuspid valves were alone engaged in the proportion of 1 per cent., and conjointly with other valves in the proportion of 10 per cent.; the pulmonary, in combination with other valves, 1 per cent.; the mitral alone, 52 per

\* Niemeyer.



cent., in combination with other valves, 85 per cent.; the aortic alone, 13 per cent., in combination with other valves, 43 per cent.\*

After birth, the contact of highly oxidized blood, and the strain to which the walls and valves are subjected in maintaining the arterial circulation, are supposed to furnish an adequate explanation of the greater proclivity of the left chambers to acute inflammatory action. On the other hand, I incline to attribute the greater susceptibility of the right chambers before birth, to the circumstance that, at this period of life, the oxidized blood from the placenta is passed in greatest part directly to the right side of the heart through the *ductus venosus* and ascending cava.

The valvular portion of the endocardium is that which is almost exclusively affected in endocarditis.

From the records of the Pathological Society of Berlin† it appears that, of 300 examples of endocarditis which had been exhibited in the three years above mentioned, inflammatory changes were confined to the valves in *every instance, except one*. The greater liability of the valves to the invasion of acute inflammatory disease may be accounted for, in part, by the concentration of the blood currents at the orifices, and the severe friction thence arising; but, chiefly by reference to the strain and tension to which the valves are subjected during ventricular systole. Taking this last mentioned circumstance into consideration, and referring only to the left side of the heart as the ordinary seat of endocarditis, an explanation may be thereby afforded of the much greater liability to attack, of the mitral than of the aortic valve. Indeed, so much greater is the liability of the mitral to acute invasion, that acute aortic valvulitis, save as a result of mechanical injury, must be regarded as exceedingly rare, and quite exceptional. Hence it may be assumed that the mitral valve is, *par excellence*, the seat of acute endocardial inflammation.

Of the pathogenesis of this affection in acute rheumatism, of which it constitutes so frequent and so grave a complication, two different theories have been propounded.

\* *Edinburgh Medical Journal*, July, 1873.

† *Loco citat.*

Doctor B. W. Richardson attributes it to the direct contact of blood impregnated with lactic acid, the product of chemical changes in the lungs during respiration, and thence conducted by the pulmonary veins to the left side of the heart. The experiments upon which he bases this theory were performed upon dogs, cats, and rabbits, and consisted in the injection of a 10 per cent. solution of lactic acid into the peritoneum.\* In these experiments the auriculo-ventricular valves were chiefly and almost exclusively engaged; the semilunar valves being only slightly thickened, but not sufficiently to interfere with their functions.

Doctor G. Reyher questions the validity of Richardson's conclusions, alleging that in several experiments performed by him on dogs, apparently healthy, a similar condition of the valves was found when death was caused by the injection of air into the jugular veins, or by poisoning with prussic acid. He urges that pathological changes in the heart-valves of dogs are of frequent occurrence idiopathically, and that the error of Richardson, and of Rauch, who, from independent experiments, arrived at the same conclusions, arose from their not having previously examined the hearts of healthy dogs. He concludes as follows: "In all the thirty-two apparently healthy dogs whose hearts were examined, the changes observed were identical with those described by Richardson and Rauch as the result of lactic acid injection.

"As Richardson and Rauch have not sufficiently avoided sources of error (owing to neglect of the examination of healthy dogs), their investigations are of no value in support of the supposed origin of endocarditis from the injection of lactic acid into the peritoneal cavity or jugular veins; consequently, that endocarditis arises from an accumulation of lactic acid in the blood remains *unproven*."†

In a communication of great practical value, Dr. Balthazar W. Foster, has recently furnished strong clinical evidence in sup-

\* *An Experimental Inquiry on Endocarditis by the Synthetical Method*, London, 1853.

† *British and Foreign Medico-Chirurgical Review*, January, 1862, from *Virchow's Archiv.*, band xx.

port of the lactic acid theory of acute rheumatism.\* Two men, suffering from diabetes mellitus, were put under the lactic acid treatment for that disease, as first recommended by Dr. Cantani, of Naples. In the first of these cases, no less than six successive invasions of acute articular rheumatism followed the use of the lactic acid in daily quantities varying from 3ij to ℞xx. The articular pain and inflammation subsided, in each instance, within twelve hours after the acid had been stopped, and they recurred with equal regularity within the same period after its use had been resumed. In the second case, one such attack followed the use of the acid, and promptly subsided when it was given up. Dr. Foster remarks: "The phenomena corresponded in all respects to those which are characteristic of acute articular rheumatism. They came on when the acid was taken, and ceased when it was discontinued. When moderate quantities of the acid were tolerated, an increase in the dose was succeeded by the painful inflammation of the joints.

"Coinciding with the development of the articular affection, was the appearance of perspiration, at first only slight, but afterwards, in the more severe attacks, copious and acid. These facts have dispelled the last lingering doubt in my mind as to the truth of the lactic acid theory of rheumatism."

According to Billroth and Weber, the blood of a person actually suffering from fever, of whatever kind, acts as an inflammatory irritant, and the endocardium is in a preeminent degree liable to inflammation from such cause.

The period of an attack of acute rheumatism at which endocarditis is most frequently manifested, is from the sixth to the ninth day, according to my observation. Fuller extends the period from the sixth to the twentieth day. Pleuritis and pneumonia, though frequently associated with pericarditis and endopericarditis, are of comparatively rare occurrence in connexion with simple endocarditis. According to Fuller's statistics, one or other form of acute pulmonary inflammation existed in the former connexions in the proportion of 1 : 1.7, and 1 : 1.4 respectively, and in the latter only in the ratio of 1 : 10.

\* *British Medical Journal*, December 23rd, 1871, and *Clinical Medicine*, 1874, p. 144.

Is pulmonary inflammation, in such cases, ever in reality the primary disease, endocarditis being secondary to, or dependent upon it? Niemeyer is of opinion that such is the case, though very rarely. I have not met with a single example of the kind, out of the many hundreds of cases of acute pulmonary inflammation which have come under my notice during the past eleven years, since I became connected as physician with a large hospital.

Owing to the fact that acute endocarditis is not fatal in the initial stage, the opportunity for determining the structural alterations attendant upon it in man has fallen to the lot of very few observers; and the doctrine held upon this subject is the result, mainly, of induction from experiment upon inferior animals.

Richardson states that when the animals experimented upon by him were examined within a period of ten hours after the introduction of a solution of lactic acid into the peritoneum, the tricuspid valve was found to be highly vascular and villous, to have lost its ordinary polish and transparency, and to have exhibited minute droplets of lymph upon the free margin. Examined at a somewhat later period, the segments were tumid, and inadequate to close the orifice, and yielded an opaque and viscid exudation on being punctured. At a still more advanced period, they were dense and thickened, but less swollen, and exhibited beneath the surface a layer of solid fibrin, and masses of the same material deposited upon the edges. Finally, they were found to have shrunk and become inadequate by retraction of their edges. With regard to the genesis of the tissue-changes which characterize the several stages of acute valvular inflammation, the first is distinguished by congestion of the *vasa vasorum*, proliferation of the connective tissue corpuscles, and consequent thickening and opacity of the valve-curtains. The second stage consists in efflorescence, or outgrowth on one of the lamellæ, by accumulation of corpuscular elements, which are liable to undergo granular metamorphosis, and subsequent disintegration and detachment under the action of the blood-current. Hence so called ulceration of the valve, if one lamella only be involved, or perforation if both lamellæ are eroded.



Upon the corpuscular outgrowth previous to disintegration, or upon the jagged edges of the ulcer or perforation caused by this process when it has actually occurred, fibrin is precipitated from the blood, forming rugosities or vegetations, which, in their turn, are liable to undergo granular metamorphosis and subsequent disintegration under the friction of the blood-stream. Virchow, however, denies entirely the occurrence of fibrinous precipitation, and attributes the primary changes of structure to corpuscular proliferation exclusively; a view which is unreservedly adopted by Niemeyer.

Other changes may follow the primary ulceration of the valves. Thus, valvular aneurism may result from the entrance of blood through the ulcerated aperture in one lamella, and the gradual detachment and distension of the other into the form of a pouch; or, a still more exceptional occurrence, veritable supuration may take place, giving rise to small abscesses, not larger than a pin's head. Even further consequences may ensue: softening and rupture of the chordæ tendineæ (Law, Niemeyer); laceration or ulceration of the endocardium at other points of its surface, leading to the "acute consecutive aneurism" of Breschet, or the "acute aneurism" of Rokitsansky, if the ventricular wall be likewise softened by extension of the inflammatory process; adhesion of the valve-segments to one another, or to the wall of the ventricle or artery as the case may be; or cohesion and fusion of the chordæ tendineæ.

Agglutination of the valves and tendinous chords by acute inflammation is, no doubt, the cause of that peculiar form of auriculo-ventricular stenosis known as "the funnel-shaped" aperture. Embolism and consequent hæmorrhagic infarction, or metastatic abscess in the spleen, the kidneys, the liver, the lungs; or, in the brain, hæmorrhagic foci (capillary apoplexy), localized anæmia and softening of the substance beyond, are possible accidents of earlier occurrence.

An immediately fatal result is, however, very seldom witnessed in acute rheumatic endo- or pericarditis. I have not met with a single example of the kind; and, of 71 cases, only 3 were immediately fatal in the experience of Dr. Peacock. In forming a prognosis, therefore, the remote consequences are mainly,

and all but exclusively, to be considered. These are of the gravest character, and may be included under the three heads of valvular obstruction, valvular inadequacy, and consecutive changes in the walls of the ventricles or auricles.

The *symptoms* of acute inflammation of the endocardium, where the muscular substance of the heart is not in some degree involved, a contingency of rare and exceptional occurrence, are by no means characteristic. This is due to the fact that they are but imperfectly localized, and, being of a general and febrile character, they are masked by those of the acute fever which constitutes the primary and the principal disease. The patient, however, not unfrequently refers to the region of the heart as the special seat of peculiar discomfort and distress. The breathing is in some degree quickened; pressure over the heart is not well borne; its action is somewhat tumultuous, and decubitus on the left side is avoided.

Even these symptoms are occasionally absent; so that the physical evidence affords the only positive information of the presence of endocarditis. This consists in the occurrence with the first sound of the heart of a murmur which, in the majority of instances, is located at the apex, and but very rarely heard at the base.

Bouillaud and Skoda lay much stress upon the extension of precordial dulness, associated with the symptoms of engorgement of the right side of the heart, as evidence of endocarditis. Such a phenomenon is, no doubt, occasionally witnessed, but only where the muscular substance of the heart is likewise deeply and seriously involved; or where, without this complication, engorgement of the right chambers arises from pulmonary emphysema with intercurrent bronchitis. Is systolic murmur always present where endocarditis exists? And, when actually present in a case of acute rheumatism, should it be regarded as proof of the existence of endocarditis? To the former question I venture to give an affirmative but qualified answer, and to the latter, a negative.

It has repeatedly happened to me, in making my daily examination of the heart in cases of acute rheumatism, to find a faint, blowing, systolic murmur at the apex, which, by many, would be

regarded rather as a muffling or prolongation of the first sound, where no symptoms or signs of cardiac complication had existed on the previous day. Most frequently this has coincided with one or more of the symptoms already mentioned as referable to the heart; but occasionally, though rarely, it has been unattended by any of these. On the following, or on the second day, this faint buzz has developed into a veritable bellows-murmur accompanying the first sound. I may add that, the adynamic murmurs of convalescence alone excepted, in no instance has it occurred to me to discover in the progress of rheumatism a fully developed murmur suddenly announced, where no trace of its nascency had previously existed; or where the coexistence of a loud pericardial frottement may not have masked it at that period of its development.

Doctor Stokes is of opinion, that in the state of typhoid debility not unfrequently associated with endocarditis, murmur may be suppressed or suspended. No doubt, in the profound debility preceding dissolution this may be the case; but, with this exception, I have not met with an example of complete suppression of murmur, where endocarditis was subsequently proved to have existed at the time, either by the development of a well pronounced murmur, or by *post mortem* evidence.

The coexistence of pericardial effusion may suppress an endocardial murmur; but the positive signs of the presence of liquid would forbid a negative diagnosis in regard to endocarditis, where murmur had not been previously detected.

Finally, the friction-sound of a concomitant pericarditis may completely mask an endocardial murmur, more especially at an early period, when the latter is not yet well pronounced. In the progress of the compound disease, the friction-sound will be definitively suppressed by cohesion of the opposed surfaces of the pericardium, whilst the valvular murmur will be heard, perhaps for the first time, in its complete intensity. Thus, the signs of pericarditis and of endocarditis proceed, as to development, in opposite directions; the former towards extinction, and the latter towards maximum intensity. But the circumstances above indicated would not warrant the inference that endocarditis had

existed without murmur; they would only show that it had attained a certain development without *audible* murmur.

The *associations* of a systolic apex-murmur in acute endocarditis, are of great importance in regard to differential diagnosis and the prospects of the patient. A murmur, *accompanied* by a first sound, and a regular though quick and weak pulse, indicates simple endocarditis; but, *replacing* the first sound, and especially if attended with a failing and irregular pulse, such a murmur should be regarded as diagnostic of endo-myocarditis. The weakness of the left ventricle, consequent upon inflammation of its muscular wall, serves to explain both the irregularity of the pulse and the suppression of the first sound; the valve element of this sound being abolished by the inflammatory thickening and softening of the mitral curtains, and the impulse element by the debility of the ventricle, consequent upon similar changes in the myocardium.

In acute inflammatory fever, the existence of an endocardial murmur must not be regarded as proof of endocarditis. It may be anæmic; and, if localized at the base of the heart, especially at the left base, it is most probably of this character, and a careful scrutiny should be instituted with a view to determining the question. The absence of *intrinsic* murmur in the carotid arteries, *i.e.*, when pressure is not made with the stethoscope sufficient to develop a murmur in these vessels; a venous hum in the jugulars; and the diminution of intensity of the endocardial murmur, or its complete suspension, on placing the patient in the sitting posture, may be regarded as proof that the murmur is anæmic. If the patient be a young female, the foregoing evidence in favour of this view is thereby strengthened; but even in the absence of this circumstance it is sufficient for the positive diagnosis of hæmic murmur. The murmur may be due to chronic valvular disease, or to this condition with secondary acute endocarditis. In the former case, the outward displacement of the apex, and the absence of symptoms of recent acute inflammation of the heart, constitute sufficient diagnostic evidence, even where the ordinary consequences of chronic inadequacy of the mitral valve do not exist; namely, pulmonary and general venous congestion, with dropsy.



The supervention of acute endocarditis on chronic valvular disease is more difficult of diagnosis; indeed, it is scarcely recognizable with certainty. The symptoms of a recent accession of acute inflammation of the heart, more especially increased embarrassment of breathing, may warrant a presumptive diagnosis; but, the physical signs being already appropriated by the chronic disease, a more positive opinion cannot be ventured. The subsequent course of the illness will, however, remove this difficulty. All the symptoms and signs of the chronic lesion are aggravated by the products of the recent affection.

Acute endocarditis is very rarely, if ever, especially in those of intemperate habits, unaccompanied by inflammation of the substance of the heart to a greater or less extent; and in proportion to the depth to which the muscular substance is involved, the symptoms assume a more formidable character. To this circumstance is due the special value of the symptoms, as distinguished from the signs, of acute endocarditis. The failure of the pulse, the restlessness and dyspnoea, the engorgement of the right chambers, rapid enlargement of the liver, congestion of the kidneys, œdema of the lungs, and the general dropsy, which occasionally supervene within a brief period, are due, not to endocarditis, but to its complication, myocarditis. The muscular substance of the heart, to the extent to which it has been implicated in the inflammatory process, will, if examined shortly after the acute attack, be found sodden and of a dead-leaf tint. At a more remote period, it exhibits the tissue-changes characteristic of fatty degeneration. Hence, whether viewed in regard to attendant symptoms, or remote consequences, the gravity of endocarditis should be measured by the extent to which the muscular substance of the heart is judged to be involved, rather than by the degree of inflammation of its lining membrane. Even in the most favourable cases, however, where the inflammation is strictly confined to the endocardium, representing but a small percentage of the total number, the remote prospect for the patient is eminently discouraging. The valves are thickened and in the first stage of disorganization, whether by morbid development of their normal elementary structures, or by deposit from the blood. Hence, retrogressive changes of structure, in-

volving proportionate incompetency of the valves, or obstruction from their morbid growth.

In the opinion of some writers, a process of tissue-change in the direction of cure may be set up; the hypertrophied connective tissue of the valves being reduced to its normal proportions by absorption, and the deposit, if any, disintegrated and detached by the blood current, to be subsequently dissolved and excreted. I cannot subscribe this doctrine, inviting though it be. The valves of the heart are not so circumstanced as to admit of this favourable change. They are, in the performance of their functions, subject to the pernicious influence of three forces operating alternately; namely, motion, friction, and tension, which are eminently calculated to maintain in them a state of constant irritation or chronic endocarditis, necessarily resulting in progressive valvular disorganization. This downward progress, once set up, may be restrained or retarded, but not arrested or reversed.

*Chronic endocarditis* is the result of antecedent acute inflammation of the endocardium, which leaves some thickening of the valves in the line of their mutual contact. This thickening, under the continued irritation arising from motion, tension, and blood-friction, increases by inflammatory outgrowth of the connective-tissue corpuscles, and subsequently, having undergone condensation and contraction, may become calcified; or, more rarely, it may pass through the regressive stages of fatty change and disintegration. Hence, extreme disfigurement, rather than destruction of the valves, is the ordinary result of chronic endocarditis.

In chronic endocarditis, the muscular substance, if the inflammation have extended to it, may be replaced by fibroid tissue; and this, being less resisting, may yield and give rise to a "true aneurism," the "chronic aneurism" of Rokitansky.

In the *treatment* of acute endocarditis, I proceed upon the principle of moderate local depletion, followed by mild but rapid mercurial action. Blood is drawn cautiously by means of leeches applied to the precordium; and from day to day this process is repeated, till the urgent symptoms of dyspnoea and precordial oppression are alleviated or subdued, the leeches

being succeeded by warm cataplasms over the heart. I have no fear of increasing the debility of the patient by these measures, deeming the prompt and complete arrest of inflammatory action, as far as this can be accomplished, the prime consideration. In view, however, of the necessarily protracted illness of the patient, I deprecate vigorous depletion, and am satisfied with leeching, being guided as to the quantity of blood drawn, and repetition of procedure, by the constitution, age, and actual strength of the patient. The pulse is usually increased in volume and diminished in rate under this treatment, whilst respiratory distress and cardiac pain or oppression are alleviated. Mercury is then administered rapidly, but in minute doses, to the point of producing its earliest results; namely, foetor, and a metallic taste. Half a grain of calomel with a grain of James' powder is given every half hour, or every hour, according to the urgency of the case, as first suggested and practised by Dr. Law; whilst a plaster, composed of half an ounce of emplastrum hydrarg. and ten grains of opium, is laid over the heart, as recommended by Dr. Beale. It will be seen that I eschew the heroic depletion of Bouillaud upon the one hand, and the copious exhibition of mercury recommended by Hope, on the other. Such extreme measures, directed to an object in my judgment unattainable, namely, the complete subdual of inflammation of the valves, or the dispersion of their endogenous outgrowths, are not warranted by even the most favourable prospects from treatment.

Whilst discountenancing the heroic line of procedure just noticed, I can by no means subscribe the sceptical doctrine of Niemeyer in regard to the alleged inefficacy of all modes of treatment. I believe that whilst it is eminently irrational to propose to one's self a complete cure of endocarditis by any medicinal agency, arrest of the process of inflammation, and restriction of its products within limits compatible with partial restoration to health, and many years of life, is attainable by the plan above proposed. Examples confirmatory of this statement will be found among the succeeding cases. To allay nervous excitement, and procure sleep, an opiate should be given at night; *e.g.*, fifteen to twenty minims of Battley's sedative, or a grain to a grain and a-half of the aqueous extract of opium. Subsequently, when the acute

symptoms have subsided, tonics should be administered; and of these I give a preference to quinine and strychnia, with or without iron, as being, in my judgment, the most efficacious. During the treatment, and long subsequently, complete repose of body and mind should be strictly enjoined. Alcoholic stimulants of the stronger kind should be prohibited; but good claret and mild bitter beer may be allowed in moderate quantity. The diet should be nutritious and concentrated; excess of liquids, whether as food or drink, being inadmissible, owing to their effect in temporarily increasing the volume of the blood, and thereby increasing the strain upon the walls and valves of the heart.

The following cases are presented as examples of endocarditis in its simple and in its complicated forms:

CASE LXXIV.—*Acute Rheumatic Endo-Myocarditis; Recovery.*

Mrs. C., aged about twenty years, was visited by me, in consultation with Mr. Cahill of Dame-street, June 20th, 1871. She had an attack of rheumatism some years ago, and a second at Christmas, 1870. Ten days previous to my visit she had a third attack; the muscles of the chest and the joints of the hands being the seat of inflammation. On the eighth day of illness, dyspnoea with cough set in, and, on the following morning, Mr. Cahill diagnosed endocarditis. When visited by me, she complained chiefly of weakness, and a feeling of oppression at the precordium. There was no swelling of any of the joints; and the muscular pains had been alleviated by the free use of a liniment of aconite and laudanum, and a flannel swathe, previously directed by Mr. Cahill. She was perspiring freely; respiration was laboured and accelerated; pulse very small and weak, 120 in the minute, but regular. There was slight cough without expectoration; the urine was scanty, and deposited lithates in large quantity. Tongue moist and coated; decubitus indifferent; no vertigo, delirium, or other head symptom. The bowels were rather relaxed, and she complained of sore throat; but there was no external swelling of the neck. The action of the heart was rather tumultuous; precordial dulness not extended; apex-beat



in the normal position, and here a very soft bellows-murmur replaced the first sound; this murmur was likewise audible in the left axilla, but not elsewhere. At the base, and in the course of the aorta, the sounds of the heart were normal, and no *frémissement* anywhere existed. The base of the right lung was dull, and yielded mucous râles. Ordered: Four leeches to precordium, to be followed by a mercurial and opiate plaster. Two grains of grey powder, and two of Dover's powder to be given every second hour; chest to be dry-cupped over right base, and then wrapped in cotton wadding. A small glass of champagne to be given every second hour. Liquid food. Chloral and opiate draught at night. I did not visit this lady subsequently, but I learned from Mr. Cahill that she made a good recovery.

Remarks: Here, as in all cases of acute endocarditis where the muscular substance of the heart is deeply engaged, the murmur of mitral reflux was not only soft and blowing, but *substitutive*; the impulse element of the first sound being entirely suspended by the softening and debility of the muscular substance of the heart produced by inflammation, and the valve element muffled by thickening, and masked by the eddy of reflux. That the implication of the heart was of recent date, and most probably coeval with the last attack of rheumatism, is shown by the normal position of the apex, and by the cardiac symptoms actually present at the date of examination.

CASE LXXV.—*Acute Rheumatic Pericarditis, succeeded four months later by Acute Endo-Myocarditis; Death. Autopsy: Hypertrophy of the Heart; General Adhesion, with Infiltration of the Pericardium; Thickening, Opacity, and Incompetence of the Mitral Valve; Fatty Transformation.*

Margaret O'B., aged thirteen years, under my care for scarlatina in December, 1872, was admitted into hospital on Easter Monday, 1873, under my colleague, Dr. Nixon, for articular rheumatism with pericarditis, from which she made a slow and incomplete recovery.

She was re-admitted on the 18th August following, in a state of debility, with dyspnoea and palpitation; the feet were slightly swollen.

About the end of August she complained of sharp pain in the region of the heart, accompanied with great tenderness, and gasping respiration. A systolic murmur, blowing and substitutive, was now audible at the apex, and faintly in the left back. The pulse became excessively weak, and also irregular and intermittent; varying as to rate from 108 to 120 in the minute. Respiration 72. There was extreme restlessness, accompanied with frequent moaning, and inability to lie down. The lips were livid, the pupils dilated, and the extremities cold. The liver was greatly enlarged. The impulse of the heart was feeble and tumultuous, and precordial dulness was much extended generally. The lower limbs became in the highest degree œdematous, and required puncturing; this was practised with great relief to the tense integument, and was followed by copious discharge of serum. Signs of pulmonary œdema and hydrothorax now appeared; the latter apparently kept in check by the free discharge of serum from the punctures in the feet and legs.

During the last week of life her condition was most pitiable; the slightest movement of her body was followed by painful palpitation, and extreme respiratory distress, accompanied with moaning. For several days preceding her death, which took place on the 15th September, there was scarcely any pulsation to be felt in the radial arteries.

The body was examined on the day of demise. There was a good deal of serum in the cavities of the pleuræ; the bases of both lungs were congested; the pericardium was universally adherent to the heart, the adventitious connective tissue which united the two surfaces, being infiltrated with lymph and serum, and fully half an inch thick at the base of the heart. It presented, in a few situations, masses of yellow plastic fibrin about a quarter of an inch in diameter, and containing areolæ filled with clear serum. Examined under the microscope, this neoplasm presented the histological characters of white fibrous tissue, and disappeared under the action of strong acetic acid. The heart, with the pericardium attached, weighed fourteen ounces; the walls of the left ventricle were thickened, and its cavity somewhat dilated; both segments of the mitral valve were greatly thickened at the edges by sub-endothelial formation, and

were incompetent; and both the valves and the adjacent endocardial lining of the ventricle were opaque. The other cavities and valves were normal. The muscular substance of the left ventricle was of a dun-brown tint; and, examined under the microscope, it nowhere exhibited a trace of striation. The fibrils were represented by parallel lines of minute oil dots, disposed in the length of the fibre. Several larger oil drops were observed free between the fibres.

I conclude that adhesion of the pericardium had existed during a period of at least four months prior to the last and fatal illness; and that this illness had consisted in general carditis, producing its specific effects in each of the tissues involved; viz., thickening and opacity, with incompetency of the mitral valve; dinginess of hue and fatty metamorphosis of the muscular substance, and fibro-serous infiltration, with softening and thickening of the pseudo-membrane between the heart and pericardium. The diagnosis of general carditis was made immediately after the access of the last acute attack, from the great respiratory and cardiac distress and failure of the pulse; but that of adhesion of the pericardium was only made presumptively. The case affords an example of serous effusion into the pericardium by infiltration of old adhesions.

#### CASE LXXVI.—*Endo-Myocarditis; Recovery.*

On the 1st January, 1873, I was summoned to visit Miss F., aged six years, of delicate constitution, and subject to anomalous pains in the limbs, supposed by the parents to be "growing pains;" but which, as they were generally associated with a febrile condition and a deposit of lithates perceptible in the urine, I inclined to regard as rheumatic.

For some days previous to my visit, she had complained more than usual of these pains, and likewise of uneasiness in the region of the heart. I found her in a feverish condition, with rapid but regular pulse, and quick breathing. There was precordial tenderness; the action of the heart was tumultuous, but the sounds were clear, and unassociated with murmur. In the course of a few days the pulse became irregular, and very weak, whilst the

first sound of the heart was replaced by a loud bellows-murmur. The breathing was very rapid, and there was a tendency to syncope, with frequent sighing and loss of sleep.

This child's condition was, for several weeks, most precarious; but she made a good recovery under the use of small doses of digitalis, iron, and spirit of nitrous ether. She has since become very fat, and is able to take moderate exercise without inconvenience; but she is readily put out of breath by rapid walking, or ascent of stairs. On examining her heart ten months later, I found a loud systolic bellows-murmur *accompanying* the first sound at the apex. The pulse was quite regular, and the general condition was that of good health.

The points of interest in this case are the irregularity of pulse, and a tendency to syncope, coincident with the development of a *substitutive* systolic murmur at the apex of the heart; and, on recovery, the restoration of the first sound *accompanied* with a murmur.

*Valvular disease of the heart* presents a most striking illustration of functional disability from mechanical impediment. The valves placed at the auriculo-ventricular orifices, and at those of the aorta and the pulmonary artery, for the purpose of imparting a definite direction to the blood-current, by preventing its retrograde movement during the systole of the ventricles and of the arteries respectively, may, by having become thickened, tuberculated, or otherwise disorganized, oppose the passage of the blood onwards in its normal course, or permit it to flow backwards into the chamber whence it had been expelled by the preceding systole. Hence, valvular diseases, functionally considered, are reducible to two generic forms, *obstructive* and *regurgitant*. Anatomically regarded, they should be classified as auriculo-ventricular, and arterial. It is more convenient, however, to designate valvular diseases specifically, as aortic, *mitral*, pulmonic, and tricuspid, respectively. If, to these latter designations, be appended those which imply the functional derangements actually present, a sufficiently precise nosological classification will have been attained. Thus regarded, all valvular diseases of the heart might be classified as follows, viz. :



Aortic	...	...	{ (a) Obstructive. (b) Regurgitant.
Mitral	...	...	{ (a) Obstructive. (b) Regurgitant.
Pulmonic	...	...	{ (a) Obstructive. (b) Regurgitant.
Tricuspid	...	...	{ (a) Obstructive. (b) Regurgitant.

But obstruction and regurgitation may be due to constriction and dilatation of the several orifices, irrespectively of the state of the valves. From direct physical evidence alone, it is difficult, and in the opinion of most writers impossible, to distinguish lesion of the orifice from lesion of the valve.

Sir W. Gull declares that "auscultation alone cannot determine whether what has been called a mitral murmur, results from organic or functional change."\*

Between these two causes of murmur, at least in regard to those dependent upon regurgitation, the differential diagnosis is, however, in a great degree practicable. It is based upon the coexistence of the sound of valve-tension, and in part upon the peculiar rhythm of the murmur, when inadequacy results from dilatation of the orifice mainly or exclusively. When the valvular element of sound is associated with murmur at any of the orifices of the heart, it may be legitimately inferred that the valves appertaining to that orifice are either sound, or but partially disorganized. If, further, the murmur be posterior to the sound in time, it may, with confidence, be affirmed that there is only a partial leakage at the orifice; and that this arises either from a trivial, and most probably a recent, lesion of the valve, or from dilatation of the orifice exclusively.

Numerous examples, confirmatory of the foregoing statement, will be found among the cases appended to the sections on aortic and mitral disease.

Valvular disease of the heart is, with the single exception of phthisis, the most prevalent form of organic disease in the United Kingdom.

On the authority of Dr. A. W. Barclay,† out of a total of 419

\* *Guy's Hospital Reports*, vol. iv., 1846, proposition 38.

† *Medico-Chirurgical Transactions*, vol. xxxi., 1848.

bodies examined in St. George's Hospital during the years 1846 and 1847, with reference to the state of the circulatory organs, 79, or a proportion of 18·8 per cent. exhibited valvular lesion of some kind. The relative proportion of aortic and mitral valve disease, single or combined, amongst these 79 cases, was as follows :

Aortic and Mitral, 36, or 45 per cent. = 8·6 per cent. of total cases.					
Aortic alone,	26,	„	33	„	= 6·2
Mitral alone,	17,	„	22	„	= 4·0
	79		100		18·8

Of 367 cases of valvular disease, collected by Dr. King Chambers from the records of St. George's Hospital, the mitral and aortic valves were affected in 121; the aortic valves alone in 107; the mitral alone in 96; mitral and tricuspid, 10; mitral aortic and tricuspid, 10; all four sets of valves, 9; tricuspid alone, 1; tricuspid and aortic, 2; aortic, mitral, and pulmonic, 2; aortic and pulmonic, 4.\*

Doctor Flint states that, in a total of 271 examples collected by him, the mitral valve was affected in 111, and the aortic in 72. In 66 of these, both the mitral and aortic were engaged. In 14 instances only were the tricuspid or pulmonic valves affected.†

My clinical records include many hundred cases of valvular disease of the heart; but, the pathological returns at my disposal, upon which alone I propose to base my calculations, furnish only 44 examples of valvular lesion. In each of these, the body was carefully examined after death by myself personally, with the aid of my clinical clerks. The following table (X.) exhibits in summary the results obtained.

\* Decennium Pathologicum, *British and Foreign Medico-Chirurgical Review*, vol. xii., 1853; and *Medical Times and Gazette*, July to December, 1852.

† *Diseases of the Heart*, second edition, 1870, p. 129.

No.	Occupation.	Male.	Female.	Age.	Obstruc.	Regurg.	Obstruc.	Regurg.	Obstruc.	Regurg.	Obstruc.	Regurg.	Right V.	Left V.	Fatty Degenerat.	Renal Dis.	Right.	Left.	Atheroma the Aorta.	Sudden De.
1	Governess	..	1	28	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	..
2	Messenger	..	..	13	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
3	House painter	..	..	30	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
4	Domestic servant	..	1	19	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
5	House painter	..	..	45	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
6	Housekeeper	..	1	48	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
7	Housekeeper	..	1	45	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
8	Peddler	..	..	68	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
9	Reduced gentleman	..	..	40	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
10	Cabinet-maker	..	..	69	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
11	Gun-smith	..	..	60	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
12	Engineer	..	..	45	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
13	Carpenter	..	..	62	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
14	Labourer	..	..	60	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
15	Tailor	..	..	70	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
16	Housekeeper	..	1	28	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
17	Gardener	..	..	53	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
18	Labourer	..	..	55	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
19	Housekeeper	..	1	39	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
20	Cabinet-maker	..	..	29	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
21	An orphan	..	1	13	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
22	Messenger	..	..	15	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
23	Car-driver	..	..	26	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
24	Discharged soldier	..	..	40	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
25	Housekeeper	..	1	44	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
26	Gas fitter	..	..	38	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
27	Labourer	..	..	38	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
28	Metal-founder	..	..	34	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
29	Brewer's carter	..	1	45	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
30	Housekeeper	..	1	25	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
31	Housekeeper	..	1	30	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
32	Domestic servant	..	..	23	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
33	Housekeeper	..	1	34	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
34	Housekeeper	..	1	50	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
35	Housekeeper	..	1	47	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
36	Messenger	..	..	14	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
37	A boy	..	..	10	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
38	Housekeeper	..	1	26	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
39	Shoemaker	..	..	22	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
40	Inmate of a reformatory	..	1	7	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
41	Housekeeper	..	1	28	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
42	Wire Worker	..	..	23	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
43	Blacksmith	..	1	36	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
44	Butler	..	..	63	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
44	TOTAL	28	16	37.3 aver.	22	17	16	25	3	4 13 dil.	1	0	24	33	20	7	36	12	22	12

An analysis of the foregoing table yields the following general results :

Total number of Cases, 44 : males 28 (= 63·63 per cent.) ; females 16 (= 36·36 per cent.) Average age, thirty-seven years and four months.

The aortic valves were engaged	...	...	...	in 26 = 59·09	% of total cases.
causing obstruction alone	...	...	...	9 = 20·45	"
regurgitation alone	...	...	...	4 = 9·09	"
both obstruction and regurgitation	...	...	...	13 = 29·54	"
The mitral valves were engaged	...	...	...	29 = 65·90	"
causing obstruction alone	...	...	...	4 = 9·09	"
regurgitation alone	...	...	...	13 = 29·54	"
obstruction and regurgitation	...	...	...	12 = 27·26	"
The tricuspid valves were engaged	...	...	...	5 = 11·36	"
causing obstruction alone	...	...	...	1 = 2·27	"
regurgitation alone	...	...	...	2 = 4·54	"
obstruction and regurgitation	...	...	...	2 = 4·54	"
The pulmonic valves were engaged	..	..	...	1 = 2·27	"
causing obstruction (a cancerous nodule) alone	...	...	...	1 = 2·27	"
Aortic obstruction was associated with mitral regurgitation	...	...	...	8 = 18·18	"
Mitral obstruction was associated with tricuspid regurgitation	...	...	...	11 = 25·00	"
Mitral obstruction and regurgitation, with aortic obstruction and regurgitation	...	...	...	2 = 4·54	"
with tricuspid obstruction	...	...	...	2 = 4·54	"
with tricuspid obstruction and regurgitation	...	...	...	1 = 2·27	"
Hypertrophy of the right ventricle was present	...	...	...	14 = 31·81	"
Hypertrophy of the left ventricle	...	...	...	33 = 75·00	"
Dilatation of the right ventricle	...	...	...	24 = 54·50	"
Dilatation of the left ventricle	...	...	...	24 = 54·50	"
Fatty degeneration of the heart was found	...	...	...	20 = 45·45	"
associated with aortic valve disease	...	...	...	14 = 31·81	"
viz., aortic obstruction	...	...	...	6 = 13·63	"
aortic regurgitation	...	...	...	3 = 6·81	"
both obstruction and regurgitation	..	...	...	5 = 11·36	"
Renal disease existed	...	...	...	7 = 15·90	"
associated with aortic valve disease	...	...	...	5 = 11·36	"
viz., aortic obstruction	...	...	...	4 = 9·09	"
aortic obstruction and regurgitation	...	...	...	1 = 2·27	"
associated with mitral valve disease	...	...	...	4 = 9·09	"
viz., mitral obstruction and regurgitation	...	...	...	2 = 4·54	"
mitral regurgitation	...	...	...	2 = 4·54	"
associated with hypertrophy of the left ventricle	...	...	...	7 = 15·90	"
of the right ventricle	...	...	...	1 = 2·27	"
of both ventricles	...	...	...	1 = 2·27	"



Renal disease ( <i>con.</i> )									
associated with dilatation of the left ventricle	...	...	...	...	in 3	=	6.81	% of total cases.	
of the right ventricle	...	...	...	...	" 4	=	9.09	"	
of both ventricles	...	...	...	...	" 2	=	4.54	"	
Thrombosis of the right ventricle existed	...	...	...	...	" 36	=	81.81	"	
of the left ventricle	...	...	...	...	" 12	=	27.26	"	
Atheroma of the aorta was present	...	...	...	...	" 22	=	50.00	"	
associated with renal disease	...	...	...	...	" 2	=	4.54	"	
with fatty degeneration	...	...	...	...	" 13	=	29.54	"	
with disease of the aortic valves	...	...	...	...	" 16	=	36.36	"	
with disease of the mitral valves	...	...	...	...	" 14	=	31.81	"	
Sudden (instantaneous) death occurred	...	...	...	...	" 12	=	27.26	"	
the valvular lesion was aortic	...	...	...	...	" 8	=	18.18	"	
viz., obstructive	...	...	...	...	" 3	=	6.81	"	
regurgitative	...	...	...	...	" 2	=	4.54	"	
obstructive and regurgitative	...	...	...	...	" 3	=	6.81	"	
The heart was fatty	...	...	...	...	" 6	=	13.63	"	
The aorta was atheromatous	...	...	...	...	" 6	=	13.63	"	
heart fatty, and aorta atheromatous	...	...	...	...	" 5	=	11.36	"	
heart fatty, aorta atheromatous, and aortic valves									
obstructive	...	...	...	...	" 3	=	6.81	"	
heart fatty, aorta atheromatous, and aortic valves									
inadequate	...	...	...	...	" 2	=	4.54	"	

The foregoing statistics may be considered to possess some value, as a contribution towards the settlement of certain debated questions in connexion with valvular disease of the heart. To these questions I shall have to revert in the progress of this chapter.

Valvular diseases of the heart are met with at every period of life; but, they are certainly more prevalent in middle age than in youth or senility. Under the title of "malformations," analogous conditions, as regards their mechanical effects, exist congenitally. These have been already considered (see p. 775). Valvular disease is likewise pretty equally apportioned between the sexes; although certain forms of it are more prevalent amongst males, and others amongst females. As to origin and rate of progress, it may commence with acute inflammation, and run its course rapidly, or slowly, according to the injury done to the mechanism of the heart in the first instance, and the circumstances in which the patient is subsequently placed, in regard to nutrition, and to physical and mental exertion. If the degree in which the valves have been structurally injured and functionally

impaired by the primary disease be but trivial, and if the mind and body be allowed adequate repose until the heart shall have adapted itself, by a gradual process of dynamical adjustment, to the altered conditions under which it is required to function, little if any inconvenience may be experienced by the patient during a life of average length and activity, as long as its nutrition is proportionately maintained. If, on the other hand, the circumstances be in all respects the opposite of those indicated, the downward progress will be rapid, in proportion as the adverse influences mentioned shall have been in operation. Irrespective of these considerations, certain habits and diathetic proclivities exercise a most deleterious influence upon valvular disease; especially alcoholic intemperance, and the gouty diathesis. But valvular disease may commence insidiously from habitual over-distension of the heart, and have made considerable progress before the patient has become sensible of its presence from notable inconvenience of any kind. This is especially true of mechanics and operatives engaged in any labour requiring great muscular effort. Habits of intemperance are capable of aggravating, in an especial manner, the evil consequences of such avocations; and, unfortunately, they are of common occurrence. The great fatigue which these men undergo, the copious perspiration caused by their labour, and the high wages they are able to earn, supply, at once, the excuse for intemperance, and the means of indulging in it.

By habitual saturation with alcohol, the blood acquires irritant properties; whilst the valves, by the repeated strain to which they are subjected, are predisposed to irritation. Under this twofold influence, sub-acute inflammation of the connective tissue of the valves, and abundant cell-growth, are prone to occur. Hence valvular thickening, obstruction, and inadequacy. Hence, likewise, corrugation, brittleness, and rupture or laceration of the valve-segments; ulceration of their surface; and deposit of fibrin by entanglement, constituting so-called vegetations.

Corvisart was of opinion that these growths are occasionally of syphilitic origin.\* Hope maintained that in many cases they proceeded from a combination of syphilis and mercury.†

\* *Opus citat.*, p. 194.

† *Opus citat.*, p. 367.

Laennec regarded them as organized fibrinous concretions, a view which Hope warmly contests, on the erroneous assumption that valvular concretions are analogous to polypi of the cavities. I have already (p. 800) shown that valvular excrescences are due primarily to cell-proliferation and upgrowth, supplemented by precipitation of fibrin upon the roughened surface of the valve. The fibrin so deposited may be promptly detached by the passing current and impacted in a distant artery, constituting an embolus; or it may undergo organization and be thereby incorporated with the valve, becoming subsequently degraded, under the influence of perverted nutrition, into one of the lower forms of tissue; viz., fat, fibro-cartilage, or calcareous nodule. Kreysig had attributed these growths to inflammation, in which opinion he was supported by Bertin and Bouillaud. Hope likewise adopted this view; adding, that he had produced them, in the course of an hour, by lacerating the pulmonic valves in an ass poisoned with woorara.\* He believed that fibrin was precipitated from the passing blood-stream, through a certain influence exercised upon it by contact with an inflamed surface. Mere roughening of the surface is, however, in his opinion, competent to produce precipitation.†

Rupture, or laceration, of the valves is the most formidable lesion to which they are liable; because of the exceptionally severe character of the symptoms to which it gives rise, and the proximate death of the patient to which it infallibly leads. No less rapidly fatal is acute valvular inadequacy by rupture of the papillary muscles or tendinous chords. I believe, however, that neither accident can occur, unless the valves or their appendages have been structurally deteriorated by antecedent disease.

Corvisart was wrong in assigning greater frequency of occurrence to disease of the aortic than of the mitral valves.‡ Laennec, whilst expressing the opposite opinion, declared that, by a singular coincidence, he had witnessed at Corvisart's clinique, within a period of three years, more numerous examples of advanced ossification of the aortic valves than had come under his notice

\* *The Diseases of the Heart and Great Vessels*, third edition, 1839, p. 370.

† *Ibid.*

‡ *Opus citat.*, section iv., p. 186.

in the following twenty years.\* No doubt, special circumstances are capable of influencing the relative frequency of certain forms of valvular disease, even amongst civil communities. This remark applies in an especial manner to disease of the aortic valves, now generally recognized as affecting commercial populations in an excessive degree. Hence, most probably it is, that amongst the population of Liverpool, Dr. Waters has met with disease of the aortic valves more frequently than any other form of valve-lesion.†

As to relative frequency, I believe valvular diseases, in the isolated form, may be arranged in the following order :

Mitral regurgitative.

Aortic obstructive.

Aortic regurgitative.

Mitral obstructive.

Tricuspid regurgitative.

Tricuspid obstructive.

Pulmonic obstructive.

Pulmonic regurgitative.

In combination, their relative frequency is as follows :

Aortic obstructive and regurgitative.

Mitral obstructive and regurgitative.

Mitral obstructive and tricuspid regurgitative.

Aortic obstructive and mitral regurgitative.

Mitral obstructive and regurgitative, with aortic obstructive and regurgitative.

Mitral obstructive and regurgitative, with tricuspid obstructive.

Mitral obstructive and regurgitative, with tricuspid obstructive and regurgitative.

Aortic obstructive and pulmonic obstructive.

Bichat committed an egregious error in denying the occurrence of valvular disease on the right side of the heart. It is, however, beyond all comparison less frequent on the right than on the left side.

In my returns (Table X.), it is as 5 : 43. Hope estimates the proportion as 1 : 16 or 20.

\* *Opus citat.*, tom. ii., p. 586.

† *Diseases of the Chest*, 1873, second edition.



According to Bertin,\* this preponderance is due to the more irritant properties of arterial as compared with venous blood, which implies that, in his opinion, it proceeds from the greater liability of the left than of the right side of the heart to the invasion of endocarditis. This doctrine cannot be questioned, and the fact may be explained quite irrespectively of the qualities of the blood, by the greater strain or tension to which the left valves are habitually subjected, as long since remarked by Corvisart.†

The heart is capable of accommodating itself, *miro modo*, to slow and progressive change of the valves, leading even to extreme disorganization, consistently with the maintenance of an adequate circulation; provided only that the duty imposed upon it be not excessive. An example strikingly illustrative of the truth of this remark is given by Dr. Stokes.‡ A gentleman of middle age and active habits, who, up to a few days before his death had enjoyed uninterrupted good health, was attacked with rigors, followed by febrile symptoms, and attended with bronchial irritation. The heart pulsated with great violence, and a systolic bellows-murmur was heard. He died suddenly, one week after the date of his first illness. The left ventricle was found greatly distended with fluid blood, and the aortic orifice was contracted to a degree which Dr. Stokes had never previously witnessed. No aperture was visible from the arterial side, but on the ventricular aspect there was a slit about four lines in length, and one line in width, "through which it was just possible to pass a fine probe." Up to his fatal illness, this gentleman had not exhibited a single symptom of cardiac disease, nor had his medical attendant the slightest suspicion that such existed.

*Disease of the aortic valves*, as judged by the preceding table (Table X.) is, by a fraction, less common than disease of the mitral valves. It is of more frequent occurrence amongst males than females, in the proportion of 21 : 5. The average age of the male subjects of the disease was forty-two years and eight

\* *Opus citat.*, p. 241.

† *Opus citat.*, p. 193.

‡ *The Diseases of the Heart and Aorta*, p. 153.

months; the youngest patient was aged seven, and the oldest seventy years. The average age of the female patients was thirty-two years; the youngest was twenty-six, and the oldest forty-four, years. Calculated in decennial periods, and grouped according to sex, the ages of these twenty-six patients were as follows:

				Males.	Females.
Under 10 years	...	...	...	1	0
10 to 20	„	...	...	1	0
20 to 30	„	...	...	3	3
30 to 40	„	...	...	5	1
40 to 50	„	...	...	3	1
50 to 60	„	...	...	2	0
60 to 70	„	...	...	5	0
70 and upwards	...	...	...	1	0
				<hr/> 21	<hr/> 5

It thus appears that disease of the aortic valves is essentially a disease of advanced life, and of the male sex. Of the 21 examples amongst males in my list, no less than 14 occurred between the ages of thirty-five and seventy years inclusive, and 10 between forty-five and seventy. In the female, the period of greatest liability is between twenty and fifty years; more than half the entire number of examples having occurred between the ages of twenty and thirty.

Doctor Milner Fothergill is of opinion, that disease of the aortic valves is more serious in the child than in the adult; because in the former it is more rapid in its progress, and usually leads to inadequacy of the valves rather than obstruction at the orifice.\* It is, no doubt, in a certain sense more serious in childhood, in consequence of the greater excitement to which the heart is liable, and the more active nutrition incidental to it at this period of life. That regurgitation rather than obstruction is the usual result of disease of the aortic valve in childhood, I agree with Dr. Milner Fothergill in holding. I believe the difference in this respect between the child and the adult is to be explained by the fact, that in the former, valvular changes are due to the incorporation of the products of acute inflammation, or cell-growth; whereas, in the adult and aged subject, they arise

\* *Lancet*, May, 1874.

from atrophic changes, by which the valves are retracted and coiled up, and so rendered not only incompetent, but obstructive also.

Disease at the orifice of the aorta, moreover, in the great majority of examples, is unassociated with rheumatism in its origin. Out of 47 cases of valvular lesion reported by Dr. Barclay,\* the aortic valves were affected in 28, 13 of which only were reputedly of rheumatic origin; of the latter, the mitral was likewise involved in no less than 12 instances.† Again, out of a total of 113 cases in which he found traces, more or less pronounced, of atheroma in the root of the aorta or in the aortic valves, 15 only yielded a history of rheumatism.‡

Of the foregoing 26 cases of aortic valve-lesion, collected by me, and made the subject of *post mortem* examination, only 2 were associated with a history of rheumatism. In 1, the disease originated with scarlatina, in 5 it was traceable to alcoholism, and in 3, to rupture of the valves by shock. I believe, however, that in these last mentioned cases, as in all similar examples, the valves were in an unhealthy condition; most probably undergoing atheromatous change at the date of the accident by which they were ruptured.

Alcoholism, then, amongst the humble classes, is *par excellence* the cause of disease of the aortic valves. Amongst the rich it is most frequently traceable to gout or the lithic acid diathesis.§ In both classes, and under the circumstances mentioned as peculiar to each, the pathological change by which the valvular lesion is accomplished is initiated by hyperæmia, or inflammatory irritation of the sub-endocardial connective tissue of the valves. Continued or repeated strain of the valves, to whatever cause due, may have a similar effect, and is likely, especially if conjoined with habits of intemperance, or with the gouty diathesis, to issue in disorganization of the aortic valves.

Doctor Clifford Allbutt|| has proved the existence of a special

\* *Medico-Chirurgical Transactions*, vol. xxxv.

† *Vide antea*, p. 398. The numbers then given as representing the author's cases of valvular lesion up to that date, now many years ago, have been since greatly augmented.

‡ *Ibid.*, vol. xxxi.

§ *Lithæmia* of Murchison.

|| *St. George's Hospital Reports*, vol. v., and *Clinical Society's Transactions*, vol. vi.

liability to disease of the aorta and its valves, amongst persons engaged in certain avocations, requiring severe, sustained, and oft repeated muscular effort, in which all the muscles of the body are simultaneously engaged, whilst the chest is subjected to compression; for example, amongst "lifters" and soldiers. He has likewise observed similar consequences, in large proportion, amongst smiths and sawyers.

Doctor Peacock declares that he has witnessed them, in many instances, in the persons of young nursemaids, who have been obliged to make great muscular efforts in lifting and carrying heavy children.

The tension to which the aorta is subjected during such efforts, is not unlikely to lead to atheroma of its walls, and aneurism or rupture of the vessel. It is, however, more frequently followed by a slow process of disorganization leading to inadequacy of the valves. Such consequences of severe muscular exertion are not, as Dr. Allbutt shows, common amongst rowers, athletes, and climbers; because, as he believes, the evil conjunction of severe, long continued, and frequently repeated muscular effort, with *compression of the chest*, is not in operation amongst them. There is much force in these reflections, and the conclusions to which they obviously lead should not be lost sight of by Parliament. I should, however, desire to include within the category contemplated by Dr. Allbutt, many classes of operatives not mentioned by him, such as rammers, mill porters, etc., who, though not subjected to thoracic constriction, are obliged to undergo labour too severe and protracted to be compatible with immunity from aortic disease. Compression of the chest during severe muscular effort is not, as far as I know, obligatory upon any body of men except soldiers. These men are not masters of their own actions; they are at the service and the disposal of the state; and to this circumstance, despite the teaching of modern science and the wealth of the country, it is due that, whilst engaged in the physical exertion necessary to qualify them for their important duties, they are forced, and without any conceivable necessity or advantage, to sacrifice their health, and shorten, by several years, the period of their lives. I have already (p. 316) urged this view, on the authority of medical officers of the greatest eminence in the



military service. Dr. Allbutt's conclusions have likewise claimed my attention (p. 485). The eminence of the writer, and the ample opportunities for the special study of this subject which a great manufacturing centre affords, entitle Dr. Allbutt's opinions to the highest respect.

I agree with Dr. Allbutt in the main, and only differ from him as to the extent to which the evils he has so ably exposed are in operation amongst the labouring portion of the community.

The legislature has justly interfered, by the Factory Acts, for the protection of youthful operatives. Why should it not, by a special Act of Parliament, restrict the period of labour of those workmen who are required, by the nature of their avocations, to make extreme and protracted muscular effort?

Atheroma is the usual cause of disease of the aorta and its valves. It was present, as already stated, in 113 out of 419 cases, or in the proportion of 27 per cent. in Dr. Barclay's returns. It is essentially a change of senility, and depends upon nutritive irritation conjoined with inadequate capacity for nutritive change.

Doctor Moxon is of opinion that it "is in continuity with arteritis, and graduates from a condition in which no inflammatory results can be found, into one in which inflammation is unmistakably present."\*

Atheroma is most frequently associated with lithæmia, and in many instances, likewise, with a granular or "gouty" condition of the kidneys. Hypertrophy of the left ventricle, to whatever cause due, leads directly to atheroma of the aorta, by subjecting its walls to severe and all but constant tension. The liability will be still greater, if, as constantly happens in gouty subjects, renal disease be likewise present; the walls of the arteries are then inadequately nourished, and arterial tension is increased by the resistance offered to the circulation in the hypertrophied arterioles. But atheroma of the aorta extends ultimately to the aortic valves; and, as Dr. Milner Fothergill has shown,† by impairing the elasticity of the aorta, it becomes a cause of imperfect coronary circulation. Hence, impairment of nutrition, fatty soften-

\* *Guy's Hospital Reports*, vol. xvi., third series, p. 431.

† *Lancet*, May 30th, 1874.

ing, and general dilatation of the heart. In the early stage of ventricular hypertrophy, whilst the walls of the ventricle and of the aorta are still structurally sound, the coronary arteries are themselves liable to become atheromatous, from the tension to which they are subjected by the elastic recoil of the aorta; a recoil, be it remembered, which is proportionate to the force of ventricular systole.

Virchow declares that chlorotic females are liable to a special form of disease of the aorta, which may lead to inadequacy and valvular disease. He has found the heart and aorta congenitally undeveloped in the subjects of chlorosis. The aorta was small in calibre, and thin. The changes in the heart were less constant; it was usually small, but in some instances hypertrophied and dilated; differences which, in his opinion, depended upon the state of nutrition, the volume of blood to be circulated, and the obstruction to be encountered in the aorta. Defective development predisposes to disease; hence, diffuse fatty degeneration of the *tunica intima* of the aorta, followed by erosions, most frequently created in the posterior wall of the aorta between the origins of the intercostal arteries.

This change in the arterial coats differs from atheroma, which commences with sclerosis of the deeper coats of the vessel, and less frequently in fatty degeneration of the *tunica media*.\*

Fatty degeneration of the heart, from the same cause, is of not unfrequent occurrence in young chlorotic females. Case 29 (p. 550) is a good example of this kind.

Acute inflammation of the aortic valves is followed by thickening, and consequent obstruction or inadequacy, or both these results, in the manner previously described (p. 800).

The changes to which these valves are liable under the influence of chronic irritation, to whatever cause due, may lead to fibro-cartilaginous thickening, or to atheromatous or calcareous transformation. The base of the valve-segments is usually the primary seat of these changes, as remarked by Corvisart; and next in order, the corpora Arantii. From these points the morbid process gradually extends over the entire valve.

Hope remarked that when the central portion of the segment

\* Abstract in *London Medical Record*, January 8th and 15th, 1873.

is solidified, the entire valve is subsequently curled up, either athwart the orifice, or against the wall of the artery. In the latter event, inadequacy of the valves, with only trivial obstruction would result, and in the former decided obstruction and incompetency.\*

Corrigan classifies the changes to which the aortic valves are liable, under four heads; viz., a reticulated condition; rupture and depression; tightening or curling up against the wall of the vessel; inadequacy of the valves by dilatation of the aorta.†

These lesions may, I think, be reduced to the five heads of

Thickening;

Foreshortening;

Depression;‡

Perforation;

Detachment.

Thickening is the result of inflammation of the valves, more or less acute. Foreshortening arises from chronic inflammation leading to abundant cell-proliferation, and subsequent atrophic changes; the valve may be simply retracted, or it may be rolled up, and at the same time elevated against the wall of the vessel, or lowered. Depression arises from over-extension of a thickened and diseased valve. One segment only was affected in the examples which came under my notice; and, except thickening, extension, and displacement by depression, whence resulted extreme incompetency, there was no observable lesion of that segment. Perforation may be the result of rupture or laceration of an unsound valve under high pressure, or of valvular aneurism; it may be likewise caused by the slower process of ulceration, a morbid change of extreme rarity. Partial detachment of one or more of the segments from the root of the vessel may occur under circumstances similar to those which cause laceration. Obstruction, or incompetency of the valves, or both, will be the result of these several changes, according to the actual position and state of the valves.

According to Dr. Peacock, the causes of incompetency of the

\* *Opus citat.*, p. 366.

† *Edinburgh Medical and Surgical Journal*, vol. xxxvii., 1832.

‡ I have never witnessed complete retroversion of the valves.

aortic valves may be classified under the following seven heads ; viz., 1. Congenital malformation ; 2. The segments cohering and not growing proportionately to the heart and aorta ; 3. Gradual stretching of the valve, and lowering of its angles of attachment to the vessel, from great and continued muscular effort ; 4. Enlargement of the orifice of the aorta ; 5. Rupture of valves, owing to inadequate support from a dilated left ventricle ; 6. Tunneling beside the valves, in cases of great and long continued obstruction at the orifice of the vessel.\*

The *symptoms* of disease at the orifice of the aorta may be conveniently grouped under the two heads of the obstructive and the regurgitative form of valve lesion. With the single exception of the pulse, it may be confidently asserted, that whilst many of these symptoms afford presumptive evidence of either lesion, they all have reference to the consecutive changes of hypertrophy, dilatation, and tissue-softening of the heart. The valve-changes, except in the case of rupture or detachment of the segments, are not directly productive of such inconvenience as to constitute a special and characteristic group of symptoms. By leading, however, to hypertrophy and dilatation of the heart, and fatty degeneration of its structure, they become indirectly the source of symptoms of the gravest augury. Manifestly, therefore, such symptoms, dissociated from physical signs, would possess no specific value as evidence of valvular lesion. Hence, the writings of the many great physicians who preceded Laennec, though full of the most valuable pathological instruction, are, in regard to diagnosis, where not positively erroneous, wanting in the point and clearness of definition which auscultation could alone confer.

Hope has clearly apprehended the distinction between the symptoms due to the valve-lesion, and the graver indications of consecutive changes in the cavities and walls of the heart. He has, however, mistaken hypertrophy for one of the conditions upon which the symptoms connected with the advanced stages of valvular disease depend. Until softening and dilatation ensue, hypertrophy, being itself a condition of compensation, will cause no greater inconvenience to the patient than that of

\* *St. Thomas's Hospital Reports*, new series, vol. ii., 1871, p. 234.



strong pulsation after exercise or excitement. It is, however, of evil augury, as a preliminary step towards the series of retrograde changes comprised in softening and dilatation. In cases of simple obstruction at the aortic orifice, the pulse, as truly remarked by Hope, exhibits no special character, unless the contraction of the outlet be extreme. In the latter contingency, the pulse will be weak; but, owing to the usual accompaniment of atheromatous change in the coats of the arteries, it will retain a certain degree of apparent fulness and volume. Irregularity and intermittence of the pulse belong, essentially, to the consecutive changes of softening and dilatation of the left ventricle, and therefore may or may not be present.

Corvisart held that the pulse is permanently irregular in all such cases; but, his statement has reference to the advanced stages, where softening and dilatation are well established.

Where inadequacy of the valves exists, however, the pulse is in the highest degree characteristic. It was described by Hope as a "jerking" pulse, or a pulse of "unfilled arteries," and by Sir Dominic Corrigan as a "vibrating" pulse. To the latter author belongs the great merit of having laid down rules by which the lesion may be identified, and of having been the first to present the entire subject in a definite scientific aspect.

In the first edition of his standard work *On Diseases of the Heart and Great Vessels*, in 1831, and therefore a year before the publication of Corrigan's paper, Hope correctly described the pulse of aortic regurgitation; but he erroneously ascribed its distinctive peculiarities to accompanying carditis, or adhesion of the pericardium. He soon, however, corrected this error, on finding the "jerking" pulse actually present where aortic regurgitation alone existed, but not before Corrigan's paper had appeared (1832). Both authors are, therefore, entitled to the merit of originality in their respective publications.

Hope was of opinion that the throbbing pulse of aortic reflux differed from that of anæmia, in being at once abrupt, and repeated at distant intervals, *celer et infrequens*. In none of Corrigan's cases was the pulse under 80 in the minute. In aortic patency, unaccompanied by obstruction, I have invariably found the pulse infrequent.

The designation of "jerking," and "collapsing," are strictly applicable to the pulse of aortic regurgitation; the former implying that, from a state of emptiness, the artery is suddenly filled with blood; and the latter, that, from a state of repletion, it is suddenly emptied. It has been likewise described as the "water hammer," and the "splashing" pulse; and, after the distinguished author of the memoir on *Permanent Patency*, "Corrigan's pulse." This pulse is characterized by suddenness of shock, rapidity and completeness of collapse, magnitude of volume, and infrequency of repetition. The abruptness of the pulse, as recognized by touch, and as shown by the verticality of the up-stroke in the sphygmographic tracings, is due to the sudden distension of a previously empty vessel. The rapid collapse of the artery depends upon the sudden emptying of the aorta through the inadequate valves, at the first moment of ventricular diastole.

M. Aran has remarked that in the progress of the disease, the pulse ceases to exhibit the "collapsing" character, owing to the circumstance, that at this period the coats of the arteries have become rigid by atheromatous change.\*

The magnitude of the pulse is accounted for by the previous dilatation of the artery, and the injection of a large volume of blood from an hypertrophied and dilated ventricle. The infrequency of pulsation is proportionate to the dilatation of the ventricle, and the volume of blood circulated at each systole.

Doctor Stokes has described,† under the designation of "steel hammer" pulse, a peculiar and characteristic pulsation of the arteries which may be witnessed in cases of acute rheumatic arthritis supervening upon chronic inadequacy of the aortic valves. The pulse is abrupt and energetic, as the rebound of a smith's hammer from the anvil; it is exhibited, however, only in the arteries adjacent to the affected joints.

Fig. LI. (p. 870) illustrates the graphic character of the pulse of simple aortic obstruction. The five subjoined tracings (Figs. XLVI. to L. inclusive) are characteristic of the pulse of aortic patency. The three latter are borrowed, by kind permission of the author, from Dr. Boileau's excellent little work.‡

\* *Archives Générales de Médecine*, tom. xv., 1842.

† *Continued Fever*, 1874, p. 244.

‡ *The Sphygmograph*, by J. P. H. Boileau, A.B., M.D., 1874.

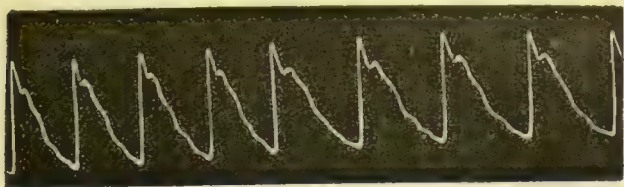


FIG. XLVI.

Aortic insufficiency. (E. G.)

This tracing indicates hypertrophy and dilatation of the left ventricle, with inadequacy of the aortic valves. Its distinctive features are, great height and verticality of up-stroke, pointed summit, well-marked tidal, and absence of dicrotic wave. Pulse regular.

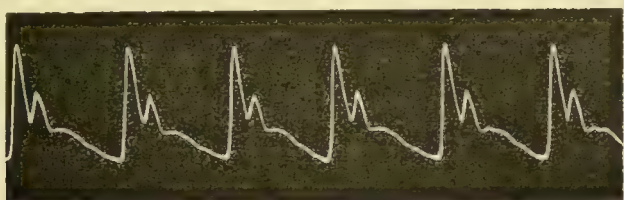


FIG. XLVII.

Aortic insufficiency. (G.)

From Dr. Grimshaw. Conditions indicated same as those in Fig. XLVI., especially the length and vertical ascent of the percussion-stroke, and the height of the tidal wave ; the former indicating great force of ventricular systole, and the latter considerable dilatation of the left ventricle.

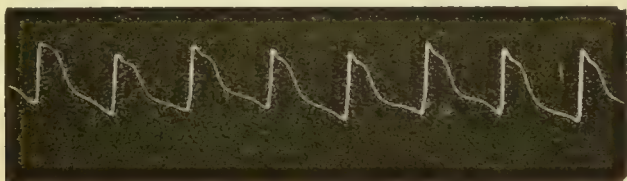


FIG. XLVIII.

Aortic insufficiency ; cardiac hypertrophy (33 ozs.) ; adherent pericardium ; ventricular dilatation. (Dr. Boileau, Fig. VII.) Pulse 110. Respiration 46.

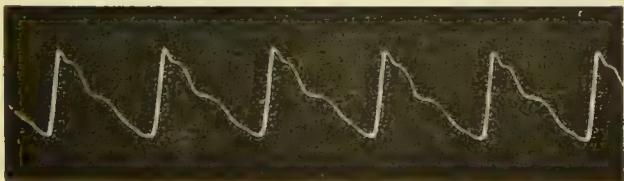


FIG. XLIX.

Aortic insufficiency. (Dr. Boileau, Fig. XXXI.)





FIG. L.

Aortic insufficiency. (Dr. Boileau, Fig. XXXII.)

Doctor Henderson maintained that the pulse is further characterized by postponement, in relation to the cardiac impulse and first sound.\* According to him, the radial pulse alternates with the impulse of the heart, "or occurs in the middle of the interval between the successive pulses of the heart." I cannot confirm this observation; indeed I am convinced it is erroneous. The arterial pulse depends, as to rhythm, upon the ventricular, not upon the aortic, systole. The characters of the pulse-tracing show that the radial pulse is in normal relationship to the ventricular systole.

A pulse possessing these characters, is, in the strictest sense, pathognomonic. It indicates, however, extreme inadequacy of the aortic valve, and is not present when a mere leakage exists at the orifice. In the early stages of the pathological change by which incompetency of the valve is produced, it must not, therefore, be looked for; and, as a guide to treatment, it is less valuable than diastolic murmur, because developed at a later period of the disease. It may, however, be a primary manifestation of valvular inadequacy, and coeval with murmur. Such is the case when it is suddenly produced by rupture, laceration, or retroversion of the valve. If, during a shock or strain, a sensation is experienced of something having given way within the chest, accompanied by sharp precordial pain, palpitation, dyspnoea with or without hæmoptysis, oppression, and faintness; and if, at the same time, right basic diastolic murmur be suddenly developed, the diagnosis of rupture, detachment, or retroversion of one or more segments of the aortic sigmoid valve may be confidently made. Fulness of volume, though usual, is by no means a constant characteristic of the pulse of aortic regurgitation;

\* *Edinburgh Medical and Surgical Journal*, vol. xlviii., 1837.



where obstruction likewise exists, it will be small and flickering. On the other hand, as already stated (p. 810), it may present a factitious fulness, even when obstruction coexists, owing to atheromatous rigidity or calcification of the arteries.

Corrigan was the first to direct attention to the remarkable symptom of "visible pulsation" of the arteries of the head, neck, and upper limbs, as characteristic of aortic patency. The throbbing of the radial and ulnar arteries is strikingly exaggerated by raising the arm above the head; it is likewise exhibited in the temporal arteries; but in minor degrees of inadequacy it does not exist in these vessels, and, when present, it is always less pronounced than in the arteries of the wrist. Visible pulsation of the arteries is not, however, pathognomonic of aortic patency; it may exist under two conditions in the absence of inadequacy of the valves; namely, when the arteries have become rigid and tortuous by atheromatous change, and when copious hæmorrhage has taken place. In the former of these conditions, visible pulsation of the superficial arteries is permanent; they are lifted visibly from their bed at each systole of the left ventricle, as the result of the elongation and straightening of the vessels, produced by their sudden distension with blood. The pulse, in such cases, would be most appropriately designated as "locomotive." In the latter condition, visible pulsation is only temporary, and coeval with insufficiency in the *volume* of the blood; but it is, nevertheless, a veritable pulse of "unfilled arteries." The differential diagnosis is, however, not difficult. Where the arteries pulsate visibly by reason of the rigid state of their walls, this condition is readily detected by the finger; it is, moreover, unassociated with sudden collapse of the vessel, which is manifestly tortuous, and the patient is also most frequently of advanced age. The principal graphic features of such a pulse (*pouls des vieillards*) as exhibited in a sphygmographic tracing; namely, great amplitude, vertical upstroke, and an extended summit, contrast strikingly with those of aortic patency. Fig. LI. illustrates these features, and may be compared with Figs. XLVI. to L. inclusive.

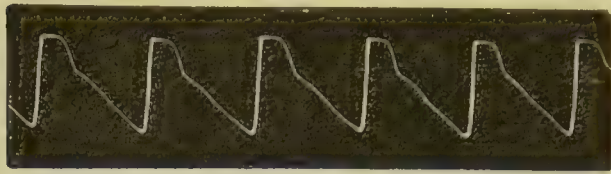


FIG. LI.

The pulse of advanced age. (Dr. Boileau, Fig. XVIII.) From a military officer, aged eighty, who had fought at Waterloo. He was in vigorous health when this tracing was taken.

Throbbing from loss of blood is associated with recent hæmorrhage. In all cases, however, the crucial test of auscultation is necessary to establish the diagnosis.

The breathing may be scarcely quickened in the state of repose, but it is readily accelerated by even moderate exercise, and is then accompanied by palpitation. Corrigan truly remarks that hæmoptysis is rare, and that œdema, when present, is always a late symptom. When, however, in the progress of the disease, the mitral valve has become inadequate by thickening of its proper structure, consequent upon the extreme and protracted tension to which it has been subjected, and by dilatation of the ventricle, pulmonary congestion and hæmoptysis, with general venous engorgement and dropsy, may ensue. But death by exhaustion, from failure of the left ventricle, usually anticipates these consequences, except when inadequacy is suddenly produced by rupture or detachment of the valve. In such contingency the left ventricle is surprised in a state of comparative unpreparedness; hence, syncope, engorgement of the lungs, and most frequently hæmoptysis, great respiratory distress, general venous stasis, cyanosis, and partial or complete failure of the radial pulse. Death is most frequently the result of such an accident in young subjects, within a few months of its occurrence, because the valves, the kidneys, and the aorta, being in such cases usually unaffected by previous disease, the left ventricle has not undergone the preparatory change of hypertrophy. But in the adult, the ventricle is most frequently in a state of partial hypertrophy when rupture of the valve occurs, owing to the previous existence of valvular, renal, or aortic disease; or perhaps of all these combined. In the three examples of this accident recorded by

Dr. Balthazer Foster,\* death followed within three months, eighteen months, and twenty-three months of the date of its occurrence, respectively. He adds, that in no well authenticated case has the occurrence of this accident been referred to a period more remote than four and a-half years previous to the death of the patient.

Four examples of rupture of the aortic valve, verified by autopsy, have come under my notice. The date of the accident was fixed with approximate accuracy by the attendant circumstances in all four cases.†

In one case, that of a woman, aged forty-four, death took place on the next day after the accident. The heart was fatty, the aorta was dilated, and the aortic valves were calcified. In the remaining three cases the patients were men, aged, respectively, thirty, thirty-four, and forty-five years; death followed the accident of rupture of the valve at intervals of two years, in the case of the two former, and nine weeks in that of the latter.

Death may follow the occurrence of rupture at a very early period also, in cases of advanced fatty degeneration of the left ventricle, as exemplified in Case 91, p. 873. In such, the ventricle is overwhelmed by the shock of the accident; being structurally unsound, it is unable to recover its equilibrium, and, owing to impairment of nutrition, it is incapable of undergoing changes of compensation.

Dilatation of the root of the aorta, by divaricating the valve-segments, deprives them of the support which, in the state of health, they afford one another by the mutual and close application of their *lunulæ*, and thus becomes a predisposing cause of rupture.

Amongst the accidental symptoms due to intercurrent congestion of the viscera are, oppression, starting from sleep, orthopnoea, angina, œdema of the lower extremities and genitals, and enlargement with tenderness of the liver. Congestion of the kidneys is indicated by albumen in the urine, but without reduction in its specific gravity, except where chronic renal disease likewise exists.

The *physical signs* are eminently characteristic, and are com-

\* *Clinical Medicine*, 1874, p. 139.

† See Cases 25, 91, 98, and 99.

prised under the three heads of, impulse, area of percussion-dulness, and murmur.

In the stages of disease at the aortic orifice, both obstructive and regurgitative, previous to retrogressive change of the substance of the heart, the cardiac impulse is strong; and in those rare cases of simple obstruction in which retrogressive tissue changes in the heart are not coeval with lesion of the valves, it is remarkably so, contrasting with the radial pulse, which is small and feeble; but subsequently to the occurrence of fatty metamorphosis the impulse is feeble; and in the later stages of the disease it is irregular, unequal, and occasionally intermittent.

In simple obstruction the point of apex-pulsation is displaced outwards, proportionately to the degree of hypertrophy of the left ventricle; in such cases, however, it rarely transgresses the nipple line by more than half an inch. But where valvular inadequacy, with or without obstruction, has been long in operation, the outward displacement of the apex is much greater, owing to the coexistence of dilatation of the ventricle. In extreme degrees of inadequacy of the aortic valve the right ventricle is also dilated, and in such cases I have found the apex-pulsation two and a-half inches to the left of the nipple line. Owing to the continued action of the reflux current upon the apex of the ventricle, that chamber is likewise elongated; hence, the point of apex-pulsation is displaced downwards, and usually occupies the sixth intercostal space; it may be detected beneath the seventh rib, and, in extreme cases, even in the seventh intercostal space.

The area of percussion-dulness is proportionate to the enlargement of the heart; but where pulmonary emphysema likewise exists, the area of dulness is proportionately restricted. The dulness extends horizontally rather than vertically; and, owing to the persistence of thoracic vibration, it yields a sensation of only partial resistance; in both these respects contrasting with pericardial effusion as previously described.\* It is further distinguishable from that due to pericardial effusion, by the circumstance that impulse is more or less distinctly perceptible over the entire area of dulness.

\* See p. 388.



Murmur is, however, the only pathognomonic sign of disease of the aortic valves. Alone, it suffices to warrant a positive diagnosis; and without it, all other signs are only presumptive. The diagnostic value of aortic murmur depends upon a correct appreciation of its rhythm, seat, and transmission. For the purpose of differential diagnosis, it is further necessary to bear in mind that the murmur may be single or double, according to the actual condition of the valves; the former indicating obstruction *or* regurgitation, as determined by the rhythm; and the latter, usually designated the "see-saw" or "to and fro" murmur, obstruction *and* regurgitation. Owing to the want of a precise distinction as to these details, the memoir of Corrigan, to whom medical science is indebted for the first complete exposition of this subject, is in some degree vitiated; but the period at which he wrote may be held as sufficient excuse for this partial defect; and the great merits of his article on "permanent patency," are scarcely diminished by it. It is necessary, however, in a modern treatise on the subject, to point out where, and in what, he has fallen into error.

Corrigan recognized only two causes of murmur in the aorta; namely, inadequacy of the valves, and aneurism. Hence have arisen certain cardinal errors in his identification of the rhythm of aortic murmurs, and in his diagnosis of the precise condition of the aortic valves. He ignored the murmur arising from obstruction at the orifice of the aorta; and when this occurred singly he regarded it as diastolic in rhythm, and as evidence, not of obstruction, but of inadequacy of the valves. The murmur is audible, according to him, in the carotid and subclavian arteries; it accompanies the "visible pulsation and diastole of these vessels," and the *frémissement* perceptible in them. A murmur answering to this description would manifestly be systolic in rhythm, and, having its seat at the orifice of the aorta, it would indicate obstruction of the valves. When two murmurs are audible in the aorta, he would regard both as arising from "great deficiency of the valves:" the first, as "from a rushing of blood up the aorta, the second from a rushing of it back into the ventricle."

The murmur indicative of obstruction at the aortic orifice is

strictly systolic in rhythm; it more frequently accompanies than replaces the first sound of the heart. When, however, the mitral and tricuspid valves are thickened or incompetent; also when the myocardium is in an advanced stage of fatty degeneration, the murmur of aortic obstruction will entirely replace the first sound.

Flint says it is oftener soft than harsh. I cannot agree in this opinion. Indeed, I have never heard a veritable aortic systolic murmur which was soft in quality; harshness is one of its proper, if not inseparable, characteristics. It is *always* transmitted into the aorta and the great arteries of the neck; but it is certainly not louder, as Flint maintains, in the right second intercostal space than at midsternum; this latter situation is, in my judgment, its point of maximum intensity. It is usually audible at the apex of the heart; and if very loud at the base, it may be heard likewise in the thoracic and abdominal aorta; but in such cases it is reinforced in the aorta by a rough and atheromatous condition of the walls of that vessel. The line of upward transmission coincides with that of the arch of the aorta, and therefore deviates somewhat to the *right* of the mesial line. Exceptions to this rule are extremely rare. Dr. Hawtrey Benson, however, has recorded one such example.\* In this case, a systolic aortic murmur was transmitted upwards and to the *left*; the arch of the aorta was found, after death, to take a similar course. When audible in the back, which is exceptional, save when it is transmitted into the descending aorta, the murmur of aortic obstruction is located in the left interscapular space. As Stokes avers, murmur of this rhythm and seat is usually associated with fatty degeneration of the heart; and, I will add, with an atheromatous condition of the aorta and principal arteries.

It may be confounded with hæmic murmur at the base of the heart; with the murmur of aneurism of the ascending portion of the arch; and with that produced by a rough and scabrous condition of the first portion of the aorta.

From hæmic murmur it may be distinguished by its harsh quality, by its being audible in the carotid and subclavian arteries in the *recumbent posture*, by the age and sex of the patient,

\* *Proceedings of Pathological Society of Dublin*, new series, vol. iv., part i., p. 34.

and, frequently, by the coexistence of atheroma of the superficial arteries. The patient is usually of middle or advanced age, and of the male sex; whereas, the subjects of hæmic murmur, not consequent upon recent hæmorrhage, are in almost all instances young females. When the murmur is purely hæmic, it is not audible in the cervical arteries in the recumbent posture, unless there be also a general arterial, or a localized subclavian murmur; and further, it is associated, in almost every instance, with the characteristic hum in the jugular veins.

The murmur of aneurism is usually localized at a higher point than the base of the heart, and is, moreover, characterized by one or more of the other signs proper to that disease. Aneurism may, however, exist at the root of the aorta, and give rise to inadequacy of the valves through dilatation of the vessel, as Corrigan showed; or it may be associated with valvular obstruction and inadequacy. Of the latter combination, a good example has been published by Dr. McAdam.\*

Finally, murmur produced by a scabrous condition of the ascending aorta, in the absence of valvular lesion, an exceedingly rare phenomenon, of which I have met with only one example (Case 62, p. 713), will be characterized by a somewhat higher seat of origin than the base of the heart, and by its *not* being transmitted into the carotid arteries. Elsewhere† I have ventured to suggest that the absence of arterial murmur in such cases may be due to the superficial, as distinguished from the axial and ordinary seat of origin of the phenomenon. It is, moreover, loudest in the curvature of the arch, or at the right second costal cartilage.

Sir Dominic Corrigan exhibited before the Pathological Society of Dublin‡ the heart of a young man, in which the root of the aorta had undergone complete osteoid transformation; it was likewise greatly dilated, and the aortic valves had been rendered thereby inadequate. During the patient's last illness, a systolic murmur of metallic quality, appropriately designated a "trumpet-bruit," was audible at the base, and in the ascending aorta and

\* *Dublin Journal of Medical Science*, vol. ix., 1836.

† *Proceedings of the Pathological Society of Dublin*, vol. v., p. ii., new ser., p. 139.

‡ See *Proceedings*, vol. ii., p. ii., new series, Feb. 1864.

carotid arteries; there was likewise a soft diastolic murmur. Sir Dominic Corrigan refers to a similar specimen which he had previously exhibited, and to another which had been brought under the notice of the Society by Dr. Banks. In this latter case, however, there had been a double "trumpet-bruit," which was found to have been caused by a "tongue of bone" projecting into the orifice of the aorta. He regards a "trumpet-bruit" as absolutely diagnostic of bony deposit in the aorta, either in the form of a "rim of bone," or a "projection or tongue of bone."

But, independently of all these causes, and in the absence of disease of the aorta or its valves, and of anæmia, systolic murmur in the aorta, propagated into the carotid and subclavian arteries, may be produced by disease of, or deposit upon, the anterior segment of the mitral valve, as exemplified in Case 31, p. 554. This rare form of lesion is most likely to be confounded with obstructive disease at the orifice of the aorta; and in the present state of science, the differential diagnosis, as between the two conditions, is, in practice, quite impossible. Theoretically, it may be supposed that, where the murmur is caused by mitral lesion, its point of maximum loudness would be somewhat below and to the left of the aortic orifice; but this is a mere hypothesis, devoid of clinical basis.

Cardiac fremitus, transmitted into the arteries of the neck, is an occasional, but by no means frequent concomitant of systolic aortic murmur of valvular origin. Fremitus may likewise attend a vascular aortic murmur; but in this association it would not, according to my observation, be perceptible in the carotid and subclavian arteries. Fremitus indicates either an extremely irregular, and, most frequently, a spiculated state of surface at the seat of its origin, or a pendulous or vibrating flap of fibrin, or of lacerated valve. I have not met with a single example of it in association with the murmur of aortic reflux, and I incline to believe that it is never so associated.

Fremitus is, therefore, intimately connected with systolic aortic murmur in origin, in rhythm, and in transmission; when it is present, murmur may be also confidently sought for; but it may be absent where murmur exists, because it requires for its production a more irregular state of surface, and more intense vibration than does the latter phenomenon.



Both the murmur of aortic obstruction, and the attendant fremitus, may be temporarily or definitively suspended by extreme debility of the left ventricle; but, so far as I have been able to observe, this occurs only at a period of the disease when there is other and indubitable evidence to show that death is close at hand.

The murmur of aortic regurgitation is most frequently diastolic in rhythm; that is to say, it occupies the period of the second sound. It may be substitutive, or accompanying; usually the latter, because the pulmonic second sound is in most cases audible in some degree at the right base. In quality it is, according to my experience, almost invariably soft and blowing, but it is occasionally attended by a musical note. It is loudest at mid-sternum, and transmitted with greatest intensity in the direction of the right apex. Dr. Foster is of opinion, that the conduction of an aortic diastolic murmur to the apex indicates regurgitation by incompetency of the *posterior* segment of the valve; whereas, conduction to the ensiform cartilage is diagnostic of inadequacy of either or both of the anterior segments.\* He adduces two most interesting cases observed by himself, in support of the opinion just stated. They are certainly of a character to lend it support; but more extended observation would be necessary to establish so important a point of differential diagnosis.

In two cases (Cases 25 and 98) I find my notes to bear in some measure upon this question. In the latter (Case 98, p. 884), the murmur, which was aortic in origin and prediastolic in rhythm, was heard at the apex of the heart, and was, owing to that circumstance, at first regarded as presystolic. On *post mortem* examination the right anterior segment was found ruptured. In the former (Case 25, p. 538), the murmur was diffused universally over the chest; and, on examination of the body, the right anterior segment was likewise that found to have been ruptured.

This question has a practical bearing. According to Dr. Foster, inadequacy of the anterior segments is of more serious import, as indicating a shorter period of survival, owing to their relationship to the coronary arteries. My attention has been too recently directed to this point, and my observations, have been

\* *Clinical Medicine*, 1874, p. 127.

too few, to warrant me in making any definite statement of opinion in relation to it. The murmur is audible, but less distinctly, in the ascending aorta, less constantly in the transverse portion of the arch, and in only a single instance (Case 25) have I heard it even faintly in the arteries of the neck. In that case the aortic valve had been ruptured, and the diastolic murmur was remarkably loud, and diffused over the entire chest. Flint asserts that this murmur is loudest in the third or fourth left intercostal space, close to the sternum. I cannot subscribe this statement.

I quite agree with Walshe in regarding the association of tactile fremitus with the murmur of aortic regurgitation, as exceedingly rare. This author declares he has met with only a single example of it.\* Sir Thomas Watson has published another.† No example of the kind has come under my observation. Considerable discrepancy of opinion exists as to the transmission of aortic diastolic murmur into the carotid and subclavian arteries. I have already stated that, in my opinion, this is in the highest degree exceptional. In reference to this point, however, I find myself at issue with very eminent authority. Sir D. Corrigan, as already mentioned, regarded carotid and subclavian murmur as one of the distinctive signs of aortic regurgitation; but I have already shown that the cases from which he drew this conclusion were, most probably, examples of the double lesion. M. Guizot, who wrote shortly after the date of Corrigan's memoir, likewise regarded carotid and subclavian murmur as a concomitant sign of aortic reflux;‡ and M. Charcelay, two years later, expressed a similar opinion.§ Flint holds|| that this murmur is "frequently but not invariably" heard over the carotids. Professor Gairdner declares that aortic diastolic murmur is propagated, "though sometimes very faintly," into the arteries of the neck.¶ M. Aran asserts that it is transmitted not only into the cervical arteries, but likewise into the brachial, crural, and tem-

\* *Diseases of the Heart*, fourth edition, 1873, p. 34.

† *London Surgical Gazette*, vol. xix.

‡ *Archives Générales de Médecine*, tom. v., 2eme série, 1834.

§ *Ibid.*, Mars, 1837.

|| *Diseases of the Heart*, second edition, 1870, p. 219.

¶ *Clinical Medicine*, 1862, p. 587.

poral arteries.\* Dr. Foster has heard this murmur in the carotid arteries, as I have at least in one instance, but he does not assert that it is usually audible† in these vessels. Independently of the teaching of actual clinical experience, it is scarcely conceivable, on theoretic grounds, that a murmur of low pitch, as that of aortic patency certainly is, should be transmitted to a point so distant, *against* the current by which it is produced.

Murmur of aortic patency may, however, be *prediastolic* or *postdiastolic* in rhythm. Examples of both these modifications of diastolic murmur in the aorta will be found amongst the cases appended to this section. I believe both are produced by leakage at the orifice, arising from defect of the valves, but in different degrees. One or two segments of the valve, or the principal portions of all three, are structurally healthy, and capable of yielding a clear second sound; but in some instances this sound is *preceded* by a faint murmur of reflux, arising from impediment to the free and rapid closure of the valves (*prediastolic*). The retardation of closure may be caused by adhesion of the segments to one another; by attachment of one of them to the wall of the aorta by means of a fibrous band; or, as in Case 98, the murmur may result from perforation and permanent depression of one of the valve-segments.

In other instances, a second sound, more or less normal in character, is immediately *succeeded* by murmur (*postdiastolic*). This may be produced either by a regurgitant axial current from retroversion of the edge of one of the valves, or from thickening and maladaptation of the segments, at the acme of aortic systole. But, *postdiastolic* aortic murmur may be likewise an indirect consequence of reduplicated second sound, owing to dilatation of the left ventricle; the aortic element of the reduplicated sound being posterior in time to the pulmonic, and subsequently being replaced by a murmur, the result of accidental rupture of the valve. Case 99, p. 886, affords a good example of this kind.

Manifestly, a similar result may follow from a slow process of disorganization, leading to incompetency of the valve, provided the left ventricle had been previously dilated, whether

\* *Archives Générales de Médecine*, tom. xv., 1842.

† *Clinical Medicine*, 1874, p. 127.

from renal disease, or atheroma of the aorta, followed by softening of the heart. Case 28 supplies an illustrative example.

I cannot agree with Flint in the opinion, that prediastolic murmur may be due to atheroma of the aorta. Diastolic aortic murmur is not necessarily permanent, as Walshe and Sanders\* allege; it may be suspended from two different causes; namely, temporary restoration of valvular competency by the entanglement of a plug of fibrin in the abnormal passage, and debility of the left ventricle from fatty degeneration, combined with a rigid and inelastic state of the aorta. Dr. Gairdner has published an example of the former kind,† and I have met with instances of the latter.

Diastolic basic murmur is, in almost every instance, due to inadequacy of the aortic valves. It may, however, arise also from regurgitation in the pulmonary artery; aneurism of the aorta immediately above the valves; patency of the ductus arteriosus, and aortic aneurism communicating with the pulmonary artery. Inadequacy of the pulmonic semilunar valves is the rarest of all valvular lesions; a murmur due to this cause would be loudest in the second intercostal space immediately to the left of the sternum, and further, it would be transmitted only downwards and towards the right apex. Aneurism springing from one of the sinuses of Valsalva is usually associated with inadequacy of the aortic valves, and is most likely to be indicated by some of the signs proper to aneurism, especially that of a *booming* quality of the attendant murmur; the latter is, moreover, systolic in rhythm, except in the very few instances in which the mouth of the aneurism is covered by the valve.

Patency of the ductus arteriosus, of which Dr. Hilton Fagge has recorded an example,‡ is indicated by murmur located considerably to the left of the sternum, and faintly transmitted downwards and to the right. Aneurism opening into the pulmonary artery is most frequently attended by murmur of systolic rhythm; and even when the murmur is diastolic, its seat of maximum intensity and line of transmission would probably suffice for the purpose of differential diagnosis.

\* *Edinburgh Medical Journal*, January, 1868.

† *British Medical Journal*, March 30th, 1872.

‡ *Guy's Hospital Reports*, 1873.



Dr. Bellingham avers that a roughened, rigid, and dilated condition of the ascending aorta, is of itself competent to produce a double murmur, which may be readily mistaken for that arising from lesion of the valves.\* The systolic murmur depends upon the rough and dilated state of the vessel; the diastolic murmur, in such a case, he would explain on the assumption, that whereas, during ventricular systole, the dilated portion of the vessel is distended with blood, during diastole it is not distended, but presents, owing to its want of resiliency, a virtual vacuum, into which the blood rushes from the carotid and subclavian arteries, under the reaction of their healthy walls. This explanation he would likewise apply to the double murmur of aneurism in this situation. But, unfortunately for this theory, *diastolic* murmur in the ascending portion of the arch of the aorta, whether in connexion with simple or aneurismal dilatation of the vessel, where the valves are not thereby rendered inadequate, and are not themselves diseased, is one of the rarest physical phenomena; whereas, both the conditions mentioned as competent to produce it are exceedingly common. I have never met with an example of diastolic murmur in a simply dilated aorta, in which inadequacy of the valves did not likewise manifestly exist. Professor Law, however, has communicated to the Pathological Society† the particulars of a case, accompanied by the morbid specimen, which he regarded as confirmatory of Dr. Bellingham's doctrine.

Jaccoud has endeavoured to show that there is a causal relationship between aortic patency and fatty degeneration of the heart, owing to the inadequacy of volume and the defective pressure of blood in the coronary arteries, which are assumed to be the necessary consequences of the former condition. There may be some ground for this statement; but, it is certain that hypertrophy is an antecedent condition to fatty change; and that fatty degeneration of the heart is no less frequently met with as an ulterior stage of hypertrophy from other causes, and notably, from simple obstruction of the aortic valves. Indeed, Dr. Stokes is of opinion, and I quite agree with him, that simple

\* *A Treatise on Diseases of the Heart*, 1857, p. 152.

† Vol. iii., p. iii., March, 1868.

aortic obstruction is all but invariably associated with fatty degeneration of the heart.

Patency of the aortic valves has been regarded as, *par excellence*, the form of organic disease of the heart in which sudden death is to be apprehended; and Walshe, basing his opinion upon eight cases which have come under his notice, goes the length of asserting that the less complex this disease, the greater is the danger of sudden death.\* Corrigan, on the other hand maintains, that in simple aortic patency death is never sudden; and under proper restriction and treatment, the patient is not only capable of leading an active life for many years, but is actually benefited by moderate exercise and labour.† He has recorded the following cases in support of this statement: A labourer, aged twenty-four, had been the subject of aortic insufficiency for seven years, during which he continued to work; this man ultimately died of acute secondary endocarditis, but not suddenly. A dispensary medical officer had laboured under aortic patency for fifteen years, and ultimately died of another affection.‡ A young man had been the subject of this disease for a period of eight years, during which he had performed with efficiency the laborious duties of a lamp-lighter, and ultimately died after a protracted illness.§ Dr. Gordon has published the following example of this kind: The patient had been for fifteen years the subject of inadequacy of the aortic valves; during the whole of this period he had worked as a mechanic, without experiencing much, if any, inconvenience from his disease; and death, which took place whilst the man was in hospital, and under Dr. Gordon's treatment, was not sudden.||

I have observed several cases of the same kind, though less striking in character; and I have arrived at the conclusion that, under ordinary circumstances, sudden death is by no means characteristic of disease of the aortic valves, obstructive or regurgitative, or obstructive and regurgitative combined.

When, with disease of the aortic valves, the case has termi-

\* *Diseases of the Heart*, fourth edition, 1873, p. 394.

† *Proceedings of the Pathological Society of Dublin*, vol. ii., part ii., new series, 1864.

‡ *Edinburgh Medical and Surgical Journal*, April, 1832.

§ *Ibid.*, March 5th, 1857.

|| *Ibid.*, vol. iii., part iii., new series, p. 276.

nated by sudden death, I believe the usual concomitant or consecutive changes of fatty degeneration of the left ventricle, and dilatation with atheroma of the aorta, have taken place ; and that these changes are mainly, if not exclusively, chargeable with the sudden death of the patient. Case 101, p. 889, may be adduced as a good example in support of this opinion.

The *treatment* of disease of the aortic valves, whether obstructive or regurgitative, should be mainly dietetic and regimenal ; having for its object the postponement of tissue degeneration of the heart and aorta, rather than the amelioration of the actual condition of the valves ; an object which is quite unattainable by any means at present known to medical science. The patient should avoid the use of alcoholic stimulants and malt drinks, beyond a very moderate daily allowance ; also fatiguing exercise or labour, as far as is practicable, especially such as involves raising the arms above the head, or so called "overhand" work ; and, whilst eschewing copious draughts of any kind of liquid, which act injuriously by increasing for the time the volume of the blood, should take plenty of nitrogenous food, of such kind as may best agree with the stomach. Animal fats, sugar, and saccharine vegetables, should be used with great moderation. The bowels should be moved at least once daily, and the skin made to act by gentle exercise, and the occasional use of the warm bath. Vegetable tonics with alkalies, such as infusion of gentian with bicarbonate of soda ; or infusion of calumba or of chiretta, with small doses of carbonate or citrate of lithia, must be administered whenever the stomach and liver, by loaded tongue and lithate deposit in the urine, give evidence of derangement. The body should be warmly covered with the view of maintaining a full and free cutaneous circulation, which acts as a natural derivative from the central organs ; and, for the opposite reason, prolonged exposure to cold should be avoided. In the warm season, however, cold bathing need not be prohibited, provided only the body be not chilled by protracted immersion. A period of a few seconds in the cold water will be quite sufficient ; and the body, whilst in it, should be briskly exercised, and afterwards rapidly dried. Sea-bathing is preferable to any other, because of its stimulant action upon the skin.

If the heart exhibit any signs of weakness, whether in the obstructive or the regurgitative form of lesion of the aortic valve, digitalis with iron, or quinine with strychnia, should be given; the former, when the action of the heart is likewise frequent and irregular, and the latter, where cardiac debility, with vertigo and imminent syncope, are the predominant characteristics. I usually prescribe the tincture of digitalis, in combination with the tincture of the perchloride of iron and spirit of nitrous ether, of each ten minims, to be taken with an ounce of infusion of quassia or of calumba three times daily, continuing the use of this medicine till the pulse-rate has been reduced, and the action of the heart has exhibited greater strength and regularity.\*

In 1832, Sir Dominic Corrigan deprecated the use of digitalis in the treatment of aortic patency, on the ground that, by slowing the action of the heart, and thereby protracting the period of diastole, it favoured the process of dilatation of the left ventricle. In 1871,† Dr. Balthazar Foster objected to it for reasons identical with those urged by Sir Dominic Corrigan. He admits, however, that digitalis may act beneficially in this disease, under circumstances indicative of over-compensation; viz., where the heart acts with violence and rapidity, and the arteries are in a state of high tension. In such a case I would prefer the application of a few leeches at midsternum, and subsequently, ten-drop doses of the tincture of aconite (B. P.), given every third hour till cardiac excitement had subsided. The tendency to syncope, which Dr. Foster and others have witnessed under the use of digitalis in this and other forms of organic disease of the heart, is, I incline to think, occasionally due, not to the drug itself, but to the mode of its administration. I believe the infusion is the most objectionable form in which it can be given, having observed unpleasant consequences, similar to those mentioned by Dr. Foster, resulting from it. But, given as the tincture, and combined with iron and ether as previously suggested, I have rarely known digitalis to cause un-

\* *Vide* p. 667.

† *British and Foreign Medico-Chirurgical Review*, No. xcv.; see also *Clinical Medicine*, 1874, p. 107.



pleasant symptoms; on the contrary, in numerous instances it has been followed by the most favourable and satisfactory results. I have, in a few instances, been under the necessity of suspending the use of digitalis owing to threatened syncope; but in the cases referred to there was fatty degeneration of the heart, in some instances with, and in others without, disease of the aortic valves. Whilst admitting that infrequency of cardiac action does not, of itself, constitute an indication for the use of digitalis, I cannot regard it in the opposite light, as prohibitory of that agent. Mere infrequency of pulse does not indicate prolongation of aortic recoil, and of influx into the left ventricle, it only shows that the rhythm of the heart is slowed by protraction of the long or diastolic pause. During this pause, blood continues to flow into the ventricles, slowly and passively, from the auricles, till, at its close, active auricular contraction takes place.

Aortic recoil is the reflex of left ventricular systole, is delivered abruptly and vigorously within the period of the second sound, and, being a purely physical phenomenon, it may be regarded as a fixed quantity, so long as the essential conditions of its development, vascular tension and elasticity, remain unaltered (see p. 91). Where incompetency of the aortic valves exists, the change of vascular tension thence arising, tends rather to abbreviate than prolong the period of aortic reaction, owing to the sudden emptying of the first portion of the vessel by reflux upon the ventricle. If, for the reasons already mentioned, quinine and strychnia be preferred, I give two grains of the former with three minims of the latter and three minims of dilute sulphuric acid, in an ounce of water sweetened with syrup of orange flowers, three times a day. Symptoms indicative of pulmonary congestion, viz., dyspnœa and feeling of thoracic oppression, cough, and hæmoptysis, should be treated by means of dry cupping, warm poulticing, and counter-irritation of the chest. Temporary engorgement of the liver and portal system, indicated by epigastric fulness and tenderness, with increased extent of hepatic dulness, constipation, flatulence, foul tongue, loathing of food and retching, and frequently also by enlargement and protrusion of the hæmorrhoidal veins, may be promptly relieved by the application of two to four leeches at the pit of the stomach,

followed by a warm fomentation or poultice; a few grains of blue pill or grey powder should also be given, and some hours subsequently, a saline aperient. During this temporary derangement of the digestive organs, thirst should be relieved by the smallest possible quantity of fluid, and the food taken should be exclusively liquid.

Dyspepsia in such cases is due, not to uræmia, as Dr. Milner Fothergill surmises,\* but to impairment or arrest of absorption in the stomach, from congestion of the portal system. I have repeatedly witnessed the symptoms previously described, where the urine was passed in full quantity, and was normal in all respects save as to deposit of lithates, and where, moreover, none of the ordinary symptoms of uræmic poisoning existed. Even assuming, as must, I believe, be now conceded, that the formation of urea is effected in the liver,† it is scarcely conceivable that the urine should not, in the cases referred to, have afforded evidence of its non-elimination, by reduction of specific gravity; and that none of the nervous symptoms usually associated with retention of urea should have been exhibited. I regard intercurrent symptoms of angina pectoris, viz., substernal pain and constriction, paroxysmal dyspnoea, and faintness, as evidence of subacute inflammation of the aorta; and I treat them by the application of leeches to the sternum, and the exhibition of small and frequent doses of mercury with chalk, and antimonial powder, combined. Two grains of each may be given every second hour. These measures rarely fail to afford prompt and complete relief.

Œdema is best treated by cupping or leeching, and subsequently poulticing the loins, with a view to relief of the renal congestion. The hot air bath may then be used with great advantage, followed by an active drastic purgative, such as half a drachm of the compound powder of jalap, or two drachms of the acid tartrate of potash in a wineglassful of infusion of senna. Digitalis, as previously directed, may be given with a view to the production of active diuresis; an object which it seldom fails to accomplish.

\* *Lancet*, May 30th, 1874.

† *Vide Functional Derangements of the Liver*, by Dr. Murchison, 8vo., 1874, *passim*.

The following cases are submitted, as examples of the various forms of lesion of the aortic valves discussed in the preceding pages. Amongst them will be also found examples of valvular inadequacy from dilatation of the root of the aorta.

CASE LXXVII.—*Hypertrophy of the Left Ventricle ; Mitral Regurgitation ; Aortic Obstruction, and Incipient Aortic Regurgitation.*

Marianne O'B., aged eighteen years, a dressmaker, admitted into hospital, June 28th, 1865. The following history was obtained from her mother. When only six years old she was taken to bathe by a female friend, and was kept in the sea fully an hour. On that evening, she was attacked with acute rheumatism of the knees and ankles ; the illness lasted five weeks, and it would seem, from the imperfect account given, that the heart was implicated. A year subsequently she had another attack of rheumatism, and sought relief in the extern department of one of our city hospitals. She waited in an outer room, where, by some oversight on the part of the attendants, she remained unobserved, and was locked up. When discovered in the evening, after several hours of confinement, she was quite exhausted from crying, and was put to bed and treated with leeching and blistering over the heart. She was also, on this occasion, treated for threatened disease of the spine. She was twelve months under treatment, and was greatly reduced in strength.

The following was her condition when she came under my notice. The pulse was not calculable at the wrist, owing to its weakness and irregularity. The pulsations of the heart were 156 in the minute, and remarkably irregular. There was orthopnoea ; the precordial dulness was much extended transversely, and the cardiac impulse, strong and heaving, was accompanied by a jarring fremitus. At the apex, a loud rasping murmur accompanied and succeeded the first sound ; no second sound was here audible. Over the lower part of the sternum, and at the base generally, both sounds were heard ; the first, accompanied by a murmur which was softer than that heard at the apex, and the second, of a partially blowing character

The basic systolic murmur was transmitted through the arch of the aorta, and into the arteries of the neck. There was dulness of the bases of both lungs; and in these situations, and also under both clavicles, crepitant râles were audible. One leech was applied over the heart, and tincture of digitalis was given in  $\mathfrak{M}$ x doses, with camphor water, every third hour.

On the following day there was evidence of slight improvement. Pulse 126, irregular; heart's action less tumultuous; face slightly bloated, especially at the eye-lids; lips and cheeks somewhat livid; teasing cough when the patient attempted to lie down.

On the 30th there was still further improvement. Pulse 108, and stronger.

The digitalis was stopped on the 2nd July; and chloric ether, with tincture of orange peel, was given in the infusion of calumba.

July 5th. Pulse 96, and distinct, but irregular. Appetite now moderately good, and patient able to sleep comfortably in the recumbent posture. Physical signs as previously noted, except that the second sound was now audible at the apex, was double at the base, and was accentuated to the left of the sternum. A systolic murmur was audible all over the front and back of chest, and was propagated into the abdominal aorta as far as the umbilicus. No visible arterial pulsation, and now scarcely any cough. The medicine last prescribed was directed to be continued, and the patient was allowed to get up. These favourable changes were fairly attributable to leeching over the heart, and digitalis given as previously mentioned.

Shortly after this date, she left hospital, but was re-admitted on the 14th; having had, on the preceding night, a syncopal attack, in which it was thought she would have died. State on re-admittance: pulse not calculable at wrist; estimated by the heart, it counted 132. Physical phenomena as last reported, except that a musical note was now audible with the first sound over the lower sternum and likewise in the course of the aorta as high as the right second costal cartilage. It was heard with three or four consecutive systoles, and then, during as many more, it ceased to be audible. Digitalis, as previously given, was resumed.



She died on the 30th August; examination of the body was not permitted. The diagnosis, however, was sufficiently clear. The musical quality of the aortic systolic murmur at the date of re-admittance was most probably due to a vibrating shred of fibrin appended to one of the segments of the aortic valve.

CASE LXXVIII.—*Visible Pulsation of Carotid and Temporal Arteries; Double Murmur at Apex and at Base of Heart, and likewise in Left Axilla; a Single Systolic Murmur in Carotids and in Descending Aorta. Death two years and nine months later. Autopsy: Pulmonary Apoplexy; Left Pleural Effusion, General Adhesion of Pericardium, and Enlargement of Heart; Hypertrophy, with Dilatation of Left Ventricle; Thickening and Inadequacy of Mitral Valve; Obstruction and Inadequacy of Aortic Valve; Diminution in Calibre of Aorta.*

John L., aged fifteen years, a messenger, admitted March 6th, 1867; had never suffered from rheumatism, but at the age of eight years had scarlatina. Subject to cough for many years, and spat blood several times; the date of the last hæmoptysis being Friday, 24th ultimo. Pulse 102, sharp, and abrupt, but not visible at wrists or in the temples. Bowels regular. No cedema. Precordial dulness extended to left. Apex-beat immediately beneath nipple, and extending over an area of one and a-half square inch in diameter.

8th. Pulse 102. A double murmur audible at apex, the systolic being the louder of the two, and of a blowing character; it is likewise audible at base, and in course of ascending aorta; but here the diastolic murmur is the louder, and *follows* the second sound, the latter being sharp, clear, and ringing over midsternum, and not accentuated over pulmonary artery. Neither murmur audible over left scapula, but both faintly so, and manifestly by transmission, in left axilla. A single systolic murmur is heard in cervical arteries. Respiration and respiratory phenomena normal; has slight cough.

April 24th. Discharged much improved in general health.

The particulars of the case at that date were noted as follows; viz., pulse of good volume, and ranging from 90 to 100. No respiratory distress. No congestion, œdema, or hepatic engorgement. Two distinct murmurs audible at midsternum, where they are likewise loudest. Of these, one is systolic in rhythm, and rough in quality; the other is postdiastolic, soft, and blowing. Both are propagated through the aortic arch, and the former, likewise, into the great cervical arteries and thoracic aorta. Second sound not intensified in pulmonary artery. Strong arterial pulsation in neck; and on right side pulsation also in internal jugular vein. Slight visible pulsation in temporal arteries, but not in radials. Treatment consisted in the use of the American wild cherry, given in form of infusion. The following note was made at this date, previously to the boy's discharge; the diagnosis being aortic valve disease, not in an advanced stage, and unassociated with the usual consequences of the affection. "Valves partially competent, reflux taking place only after the act of closure, and giving rise to a murmur, postdiastolic in time, *i. e.*, immediately succeeding the second sound, which was sharp and clear; thus indicating a condition of valves in which their *tensive* properties were not impaired, but their edges were probably tuberculated, and incompetent by want of coaptation."

On the 9th November, 1869, this patient, now a grown young man, was re-admitted for rheumatic pains in several joints. There was a visible pulsation in the carotid, temporal, and brachial arteries. Pulse at wrist sharp and collapsing, and arhythmically irregular. Apex-beat in nipple line; and here, a fremitus was perceptible. Here, likewise, a double impulse was felt, and a substitutive systolic bellows-murmur was audible. At base a double murmur was heard; viz., a systolic, which was likewise transmitted into the carotids, and a soft and blowing murmur *succeeding* a sharp second sound.

December 29th. After a fortnight's absence from hospital duty, I found him much emaciated, and expectorating blood-stained mucus. Respiration embarrassed. Precordial dulness extended. He complained of an overpowering sense of oppression across the chest. A blister was applied to the front of the

chest, and some antispasmodic medicine was given. On the following day he felt much relieved. Pulsation was then slightly visible in the radial arteries, and the second murmur at the base was rather diastolic and substitutive, than postdiastolic in rhythm. No cedema.

January 4th, 1870. Over border of left costal cartilages, in nipple line, and to a distance of two inches around this point, as likewise over entire epigastrium, a loud metallic tinkle was heard synchronously with the double impulse of the heart. It was loudest in the first mentioned situation, and was audible during expiration only; the descent of the diaphragm and inflation of the lung seeming to suspend or mask it, probably by separating the pericardium and stomach.

12th. He died suddenly, and apparently of syncope, during an effort of vomiting.

Examination of the body twelve hours after death. A few nodules of extravasated blood were found in the lungs. The left lung was compressed by serous effusion into the pleura, and its surface was opaque and corrugated. The pericardium was thickened and universally adherent to the heart; the false membrane by which it was attached, was two lines thick, and presented a blood stain posteriorly. The heart was greatly magnified and elongated, and, with half an inch of the great vessels attached, it weighed twenty-eight and a-half ounces. Right chambers normal. In the right ventricle there was a thick flake of yellow fibrin connected with the tricuspid valve, and extending, in the form of a flattened cylinder, into the pulmonary artery. Left auricle thin; appendix bound down to surface of auricle by adhesion. Anterior right segment of mitral valve presented three wart-like excrescences on the auricular aspect of the free margin, and in the vicinity of these, the valve was slightly thickened. The remainder of this valve, as likewise the entire of the posterior segment, was sound. The walls of the left ventricle were an inch thick at the base, and its cavity was much dilated and elongated. A thin flake of fibrin adhered to the septum. The aorta was reduced in calibre to the size of the middle finger. Its lining membrane was of a scarlet tint, and its valve-segments were shrivelled or rolled up at the free edge; but structurally

they were apparently healthy. When approximated by means of water poured into the aorta, they left a large opening in the axis of the passage, through which the water rapidly flowed into the ventricle. The aorta and its primary branches contained some dark blood-clot.

In reference to this case, I wish only to observe upon; (a) the upward displacement of the point of apex-pulsation, in connexion with general adhesion of the pericardium; (b) the existence of a basic *postdiastolic* murmur, where incompetence, without structural disease of the aortic valve existed, and the subsequent conversion of this into a veritable diastolic murmur, at a time when, presumably, the valve was beginning to undergo structural change; (c) the absence of accentuation of the second sound in the pulmonary artery, and of audible murmur at the left scapula, where veritable mitral reflux existed; (d) the existence of only a systolic murmur in the carotids, where a double murmur was audible at the base, and in the arch of the aorta; and, (e) the existence, at the precordium, of a metallic tinkle communicated from an inflated stomach, and assuming the rhythm of the double cardiac impulse.

CASE LXXIX.—*Anasarca and Albuminuria; Extension of Precordial Dulness Transversely; Feeble Impulse of Heart, and Basic Systolic Murmur transmitted into Aorta and Carotids; Death. Autopsy: Effusion into the Pleuræ and Pericardium; Hypertrophy and Dilatation of Left Ventricle; Obstruction and Incompetency of Aortic Valve; Renal Disease.*

James A., aged twenty-six years, a car-driver, admitted June 4th, 1867. Two months previously he had a chill, which was followed by cough and expectoration. At the date of admittance he was remarkably pallid, the lower limbs and trunk were swollen, and there was cough with expectoration of rust-coloured mucus. Respiration 48. Pulse 120, and dicrotic. Throbbing of carotids. Slight congestion of lips. The area of precordial dulness was increased transversely. No perceptible cardiac impulse. At the apex of the heart, a feeble and distant systolic murmur was audible, and the second sound was faint, but other-



wise normal. Over midsternum, a loud systolic bellows-murmur was heard; it was likewise audible in the arch of the aorta, and faintly in the carotids. Crepitant râles were audible all over the right side. Urine passed in small quantity, 1·015 in sp. gr., loaded with albumen, and containing numerous red-blood corpuscles, and a few hyaline tube-casts to which some epithelium still adhered.

9th. Patient pale and somnolent. Precordial dulness increased in extent. Heart-sounds obscure, and no murmur to be heard. Comparative dulness at postero-inferior portion of right side, where crepitant râles are audible.

10th. Died early this morning. *Post mortem* examination of body *vesperi*. Copious effusion of serum into both pleuræ and pericardium. Heart greatly enlarged transversely, and by hypertrophy of the left ventricle exclusively; right chambers normal, and containing some dark coagulated blood; left ventricle much dilated and thickened. The mitral orifice and valves were normal and competent. All three segments of the aortic valve presented, at their free edge, a crop of rough granular vegetations, which extended to a considerable distance on the ventricular surface of the valves, so as necessarily to obstruct the exit of blood from the ventricle. The left anterior segment presented a rent or opening, as large as a pea, near its free margin. The edges of this rent hung into the ventricle, and were coated with granular lymph, forming a tubular passage about half an inch in length, which permitted regurgitation, and at the same time, by its prominent margins, obstructed the orifice of the artery. The edges of the valves, moreover, hung loosely into the ventricle, and permitted free regurgitation. The substance of the heart was firm. The aorta was healthy. Liver and spleen greatly enlarged and congested. Kidneys enlarged, their cortex thickened, and presenting a light fatty appearance. By an oversight they were not examined microscopically. The lungs were examined, but no note was made of their condition.

CASE LXXX.—*Rheumatic Fever; Œdema; Weak and Intermittent Pulse; Paroxysms of Violent Palpitation and Cardiac Asthma, with Extreme Rapidity of Pulse; Jugular Pulsation; Enlargement of Liver, and Ascites; Suppression of Second Sound of Heart; Systolic Murmur at Apex, and Double Murmur at the Base of the Heart.*

Owen G., aged twenty-three years, a farmer, admitted into hospital, September 4th, 1867. Has worked hard as an agricultural labourer. Eight years since he had measles, and two years subsequently, rheumatic fever after a severe wetting. Shortly after the last mentioned illness he began to suffer from palpitation and shortness of breath on exertion. He likewise had occasionally œdema of the feet, which passed off with critical diarrhœa and diuresis.

When admitted, he was suffering from general œdema, dyspnoea, and palpitation. The urine was moderate in quantity, 1.025 in sp. gr., acid, and free from albumen and sugar. Copious diaphoresis. The hair of the head was erect, and of a spinous character. Precordial dulness extended horizontally from the left nipple line to the right of the mesial line of the sternum, its vertical extent being normal. A loud systolic bellows-murmur was heard below the nipple, and likewise, but less distinctly, over the base, and at the xiphoid cartilage. A second sound was nowhere audible. The supraclavicular fossæ were obliterated, and on the right side there was visible pulsation of the external jugular vein, which was synchronous with the impulse of the heart, and more distinct with inspiration than with expiration. The chest was prominent on both sides, and on the right there was considerable dulness below the clavicle, and all over this side loud râles were audible. Over the left side, on which he seldom lay, respiration was puerile. The liver was enlarged, and tender to pressure; and there was some liquid in the peritoneum. The radial pulse was slightly visible, weak, and intermittent. The intermission recurred at unequal intervals; thus, it was noted after six, eight, nine, and fourteen pulsations, respectively. On the 9th September it occurred four times in succession, after

seven, eight, and nine beats respectively; then after thirty-two beats; and next, four times successively after fourteen beats.

R Infusi pruni Virginianæ, ʒviiss; Spirit. æther. nitros., ʒiv. A tablespoonful to be taken every fourth hour. To have saline aperients as required; nutritious diet; and a moderate allowance of wine.

On making my morning visit on the 10th September, I found him suffering from an aggravated paroxysm of cardiac asthma; the heart was acting tumultuously, and at the rate of 198 pulsations in the minute; respiration 48, and the body bathed in perspiration. No cardiac murmur was then audible. This paroxysm occurred without any assignable provocation. To have immediately a draught composed of Hoffman's anodyne, ʒss; Battley's sedative, ʒj; and camphor water, ʒj.

On the following morning (11th) the pulse was only 98, regular, and without intermission.

16th. It was noted that since last report he had had one paroxysm, but of a less severe character than the first. The pulse had varied from 96 to 108, and been occasionally free from intermission. The sedative draught last mentioned had been continued, and a pill had been given daily, consisting of equal parts of blue pill, dried soda, and compound rhubarb pill; and on that morning, owing to the existence of unusual hepatic tenderness, a single leech was applied over the liver, and subsequently a warm poultice.

17th. 11 A.M. I found the patient again in a paroxysm. Pulse 198, and respiration 54. No murmur detectable. Sedative draught repeated.

18th. Complained of "swimming" in the head when he dozed. Pupils small; pulse 96, regular, and not intermittent. Tincture of hops (ʒj) was substituted for opium in the draught.

23rd. 10 A.M. Patient sitting up in bed, face flushed, and body bathed in perspiration. Pulse failing, and not calculable at the wrist; but registered by the heart, it was 210 in the minute. The right external jugular vein and its tributaries were greatly distended. To have former sedative, but without opium.

He passed a good night on the 24th, and on the following morning the pulse was 108 and regular. Considerable cedema of

left hand and forearm, notwithstanding that decubitus is usually *dexter*. No second sound audible.

27th. A double murmur was audible at midsternum, to the left of the median line; and a faint but rough second sound was heard in the course of the aorta. Pulse 96, steady, and regular. Much cedema of left hand and forearm.

October 5th. Patient flushed, and perspiring. Pulse 192; respiration 36. This paroxysm, which came on during sleep, was of brief duration. He passed a good night, and on the following morning (6th) the pulse was only 84.

November 8th. 11 A.M. Pulse 210, counted by the heart; respiration 48, and accompanied with great dyspnoea and inability to speak; face flushed and perspiring. The entire chest was visibly shaken by the violent action of the heart.

On the evening of this day, the patient abruptly left hospital, and I have not since heard of him.

The details of this case, even in the absence of *post mortem* evidence, are given at great length, because of the typical character of the cardiac asthma, and the extreme rapidity of pulse which it exhibits. I have met with only two cases, of which this was the first, in which the rate of cardiac pulsation reached 200; in this instance, on two different occasions, it exceeded that number by 10 beats. The diagnosis implied in the title of the case is, I venture to think, warranted by the symptoms and signs exhibited.

CASE LXXXI.—*Double Basic Murmur, remarkably Loud and extensively Transmitted; History of Sudden Occurrence of Cardiac Lesion from Strain. Diagnosis: Yielding of Segmentation of Aortic Valve previously unsound.*

Richard W., aged thirty-six years, a labourer of intemperate habits, was admitted, December 29th, 1868. Had been for some time subject to cough; and six days prior to admittance, whilst carrying two buckets full of liquid food to cattle, he felt a sudden yielding in the region of the heart, which was followed by a buzzing noise distinctly audible to himself. He felt no pain in the chest, and had no paroxysm of dyspnoea either then or



since. There has not been hæmoptysis. The buzzing noise in the chest has continued up to the present, and in bed it has been particularly loud and troublesome.

The appearance of the man indicates good health. There is, however, shortness of breath with orthopnoea, but no œdema. Pulse in erect posture 108, regular, and equal on the opposite sides, but visible; in the recumbent posture it is 84. Chest universally resonant, and respiration natural.

In the erect posture a fremitus is perceived on placing the hand over the precordium; the seat of this fremitus is basic. Precordial dulness normal in extent. Apex beat in fifth intercostal space and in nipple line, and perceptible over an area of one inch in diameter. Here both sounds are accompanied by murmur, manifestly transmitted; the first, or systolic, soft and blowing, and the diastolic, jarring and faintly musical.

At midsternum these murmurs attain their maximum intensity, and replace the normal sounds of the heart; the systolic, loud and blowing, and the diastolic, loud, buzzing, and musical. Both murmurs, retaining the characters just described, are diffused over the entire front of the chest, but with a loudness progressively diminishing in proportion to the distance from midsternum. The diastolic murmur is likewise audible in the carotid and brachial arteries, in the abdominal aorta and iliac arteries, and faintly also over the lumbar spine. In the recumbent posture, and after repose, it loses its musical character, and the fremitus is then not perceptible at the precordium.

After a short residence in hospital, this man returned to his employment, somewhat improved by rest. I have not since heard of him. The features of special interest in the case are: the absence of sub-sternal pain, dyspnoea, and hæmoptysis at the time of the accident, assuming it to have been of the character diagnosed; and the transmission through the principal arteries of the second order, and to a great distance, of an aortic *diastolic* murmur. In both these respects the case is, in my experience, unique.

CASE LXXXII.—*Repeated Attacks of Acute Rheumatism; Double Cardiac Impulse, and Double Murmur of Aortic Origin; a Systolic Murmur audible in the Arteries of the Neck, and a Diastolic Murmur in the Thoracic and Abdominal Aorta.*

Thomas B., aged twenty-two years, an accountant, visited hospital as an extern, January 30th, 1874. Had acute rheumatism seven years previously, and a similar attack each succeeding winter since that date. Has smoked and drunk to excess, but for the last six months has been temperate in both these respects. Has not had hæmoptysis. Is pale and nervous; breathing natural. Pulse 108, and remarkably variable as to rate independently of physical effort, and visible in carotid, temporal, and radial arteries. Can lie on left side. There is strong, diffused, and double cardiac impulse; no localized apex beat. At midsternum there is a double murmur, the systolic element of which is of a blowing character, and ends with a sharp and clear valve-click, the diastolic being much louder, and also blowing. Both murmurs are audible all over front of chest, and distinctly below and outside left nipple line. The systolic murmur alone is heard in the carotid arteries, and the diastolic only, but very distinctly, along the dorsal and lumbar spine. To have tincture of the perchloride of iron, and spirit of nitrous ether, of each ℥x, with ℥v of tincture of digitalis, in an ounce of infusion of quassia, thrice daily.

The diagnosis is given in the title of the case, and no comment upon it is necessary; except that it exemplifies still further the truth, so often previously insisted upon, that whilst aortic murmurs of diastolic rhythm are extensively transmitted through the main trunk of that vessel, they are *not* audible in its primary branches; whilst aortic systolic murmurs of valvular origin *always* are audible in these vessels. Case 81 affords a notable exception to the former statement. The following case still further illustrates the preceding remarks.

CASE LXXXIII.—*Double Aortic, and Systolic Mitral Murmur. Diagnosis: Aortic Obstruction and Reflux, and Mitral Regurgitation.*

James O'B., labourer, aged thirty years, seen as an extern,

May 28th, 1869. Has been three years in the army, but never in active service, and retired three years ago. Has had palpitation for the last ten years; but his health failed only at Christmas last. Pulse 84. Visible pulsation of carotid, temporal, brachial, and radial arteries. Respiration embarrassed. Decubitus *dexter*. Cough, with copious expectoration. Engorgement of both lungs posteriorly. No œdema anywhere. Cardiac impulse heaving, and apex-pulsation diffused.

A double murmur was audible at the base and in the ascending portion of the arch of the aorta, the diastolic being the louder, and not audible in the neck; whilst the systolic murmur, which became fainter in the ascending portion of the arch, was exaggerated in the carotids, and was there attended with fremitus.

Immediately to the right of a vertical line two inches inside the left nipple, the systolic murmur was faint; but to the left of that line it was louder, and thence to the site of apex-pulsation in the nipple line and sixth intercostal space, it gradually increased in intensity. The second sound in the pulmonary artery was not accentuated, and no murmur was audible in the back. To have tincture of the perchloride of iron, chloric ether, and ipecacuanha wine in camphor water.

The following case is remarkable in regard to its duration, its apparent gravity being considered.

CASE LXXXIV.—*Dyspnœa ; General Venous Congestion and Œdema ; Extreme Weakness and Irregularity of Pulse ; Mitral and Aortic Regurgitation.*

Bridget M., a domestic servant, aged twenty-six years, admitted into hospital in the early part of 1868, and seen again as an extern on the 26th October in that year. Respiration much embarrassed after exertion, and oppressed even when she is at rest. Pulse remarkably weak and irregular, and not calculable at the wrist. Action of heart irregular and intermittent. Œdema of feet, and congestion of lips. At the apex of the heart there was a systolic murmur, and at the right base a soft dia-

stolic murmur replaced the second sound, which was audible only in the pulmonary artery.

This poor girl was re-admitted in July, 1872, in the same condition; she left, relieved, after a month's rest and tonic treatment.

CASE LXXXV.—*Systolic Murmur at Apex; Double Murmur at Base; Systolic Basic Murmur extensively transmitted through Arteries. Diagnosis: Mitral Regurgitation; Aortic Obstruction and Regurgitation.*

Henry H., aged thirty-six years, a labourer, admitted July 1st, 1867. Health reported good up to six months ago, when, without having been ill, or having sustained injury of any kind, he began to suffer from pain in the left side, and lower part of the back. Two months subsequently he observed a swelling in the left side of the abdomen, and in the site of this swelling he had occasionally pain of a spasmodic character. Urine high coloured, and depositing amorphous lithates. Pulse 68, and "collapsing." Visible pulsation in carotid, radial, and femoral arteries. Strong pulsation of abdominal aorta, accompanied by a purring tremor and a systolic jarring bruit. A systolic bruit of a somewhat rough character existed at the apex of the heart, which pulsated in the sixth intercostal space, one and a-half inch outside the nipple line; it was audible over an area measuring one and a-half inch transversely, by one inch vertically. This murmur was likewise heard in the left axilla and left back. At the apex, the second sound was not distinct.

At midsternum, a systolic and a diastolic murmur existed; both were transmitted in the course of the ascending aorta, and were loud, soft, and blowing, but the diastolic was the louder of the two, and superseded the second sound. The systolic murmur was propagated into the carotids, abdominal aorta, and femoral arteries. Second sound not exaggerated in the pulmonary artery. No dyspnoea. Discharged somewhat relieved, July 14th.



CASE LXXXVI. — *Systolic and Diastolic Basic Murmur, the former accompanying, the latter substitutive; Reduplication of Second Sound. Diagnosis: Obstruction and Regurgitation at Aortic Orifice, and Considerable Dilatation of Left Ventricle.*

James H., a stucco-plasterer, temperate, aged forty-four years, admitted into hospital, September 27th, 1869. Has never had rheumatism, but has had flying pains in various parts of the body. During the last six months has been weak, and often, within that time, has nearly fallen down in the streets from faintness. Sight began to fail, especially in the right eye, three weeks ago, and is now very much impaired; the right pupil is clear, but scarcely responds to light. Patient remarkably pale. Respiration tranquil; decubitus indifferent. Pulse 78, bounding and visible. No œdema. Apex of heart pulsates half an inch outside nipple line, in the fifth intercostal space, and over an area of one inch in diameter. Here both cardiac sounds are obscure, a faint transmitted murmur accompanying the second sound. At midsternum, and also in the course of the ascending aorta, a double murmur is heard, the systolic being the less distinct, and accompanying, whilst the diastolic murmur is loud, blowing, and substitutive. The former is transmitted into the carotid arteries, but the latter is not. No murmur audible in back. Second sound normal as to quality, and not accentuated in the pulmonary artery; it is double throughout the precordium, the first element being sharp and clicking, the second element represented, as already stated, by a loud bellows-murmur, which is most distinct at base, and masked and prolonged at apex. R. Tinct. ferri perchlorid., æther. chloric., aa. ℥x tertîa, q. q. horâ s.

On the 9th October, which is the date of latest report, the second sound was not anywhere audible. The man left hospital soon afterwards.

The coexistence in this case, as in others to be afterwards noticed, of aortic regurgitation with double second sound, enabled me to identify with certainty the aortic and the pulmonic elements of that sound, respectively. The former, which was

posterior in time, was represented by a murmur; whilst the latter was sharp and clear. I believe the relative position in time of the two factors of the second sound, in this and all similar cases, may be accounted for by the existence of dilatation of the left ventricle, owing to which the evacuation of that chamber, and therefore the reaction of the aorta, are necessarily postponed.

If this view be correct, it follows that the existence of double second sound, in connexion with the murmur of aortic reflux; indicates dilatation of the left ventricle.

CASE LXXXVII.—*Rheumatism, Cough, and Hæmoptysis; Orthopnoea and Paroxysmal Dyspnoea; Congestion of Liver, and General Œdema; Visible Pulsation of Arteries; Increased area of Precordial Dulness; Feeble Impulse; Double Murmur at Base, and a Single Musical Systolic Murmur in Aorta and Arteries of Neck, and likewise audible along the Spine; Gangrene of Feet; Hæmoptysis; Death. Autopsy: Heart greatly Enlarged, and Laden with Fat; Aorta Dilated and Atheromatous, and its Valves Incompetent by Dilatation of the Vessel, but also slightly Diseased and presenting Obstruction to Exit of Blood; other Valves healthy; Right Ventricle Thickened, but not Dilated; Left Ventricle Dilated and Thickened.*

John C., aged forty years, admitted November 16th, 1869. Has served twenty-one years as a soldier, ten of which were passed in India. Had an attack of rheumatism eight years ago whilst in camp at Shorncliffe, and has had cough for the last twelve months. Left the army last May. A fortnight ago he spat blood for two days, and about the same time his feet began to swell. For the last four months he has not been able to lie down, owing to the immediate occurrence of urgent dyspnoea on his attempting to do so; and for the latter half of that period he has been subject to paroxysms of dyspnoea, coming on suddenly whilst he lies quietly propped up, and obliging him to start out of bed to avoid being suffocated.

State on admittance: feet, legs, and thighs greatly swollen,

and very tense, but pale and waxy-looking. Scrotum also cedematous. Toes congested; face of an olive tint, but not jaundiced. Liver enlarged and tender. Pulse 96, and visible in the carotid, temporal, and radial arteries. Precordial dulness much extended, but cardiac impulse diffused and feeble; no distinct apex-pulsation to be felt. At base, a systolic and a diastolic murmur existed; the former musical, and transmitted through the aorta to its bifurcation, and likewise into the carotid and subclavian arteries; it was audible along the dorsal and lumbar spine, and, by transmission, at the apex. The diastolic murmur was soft, and confined to the base and the ascending aorta. Congestive enlargement and tenderness of the liver. To have an aperient, and afterwards a mixture of tincture of the perchloride of iron and chloric ether, in camphor water. To have, at night, a sedative, composed of Hoffman's anodyne and liquor of hydrochlorate of morphia. Feet and legs punctured, and yielded a copious discharge of serum.

26th. Lower limbs now enormously swollen, red, and tender, and on dorsum of right foot, incipient bullæ, indicative of proximate gangrene. Interval between cardiac impulse and radial pulse quite distinct, the pulse-rate being 96. The stroke of the radial artery coincided with the second sound of the heart; whereas, in the absence of organic disease of the heart and aorta, as I have ascertained by experiment, this does not occur till the rate of cardiac pulsation has attained to 126 in the minute.\* Patient insists on having the window opposite his bed kept open day and night; and to admit of this being done without danger to the other patients, his bed was removed to a window at the end of the ward, and there enclosed on three sides by means of screens. Is spitting blood.

28th. Died this morning. The feet had previously assumed a dark chocolate hue, and several gangrenous bullæ had formed upon them. For some time before death the patient had ceased to crave for fresh air, and desired that the window should be closed.

\* *Vide* paper "On the Rhythm of the Heart," Proceedings of the Medical Society of the College of Physicians, *Dublin Quarterly Journal of Medical Science*, August, 1864. This is the only example of veritable postponement of the radial pulse which I have met with. See p. 832.

On the following day, the heart, which was the only organ I could procure for examination, was carefully inspected. Emptied of blood, and with an inch and a-half of the aorta and pulmonary artery attached, it weighed twenty-seven ounces; it was of a globular figure; the apex was blunted, and the base was extensively and deeply covered with fat. The aorta was dilated above the valves, and readily admitted three fingers introduced edge-wise. The valves were incompetent, water poured into the aorta readily flowing into the left ventricle. The line of union of two of the valves was hard and cartilaginous, and offered an obstacle to the efflux of blood from the ventricle. Otherwise, the valves were healthy. The lining membrane of the aorta presented several yellow patches of incipient atheromatous change. All the other valves were normal and competent. Right ventricle somewhat thickened, but not dilated. Left ventricle dilated, and three-fourths of an inch thick at base and centre. Muscular structure healthy.

CASE LXXXVIII.—*Scarlatina and Aortic Valvulitis; Extensively transmitted Aortic Systolic Murmur. Diagnosis: Aortic Obstruction, Embolism, and Recurrent Right Hemiplegia.*

Thomas B., aged seven years and a-half, a fine healthy looking boy, was admitted into the Mater Misericordiæ Hospital, under my care, on the 5th of February, 1874.

Had scarlatina three years previously. Three months prior to date of admittance, he complained of "pain in the stomach," which quickly passed away; nine weeks later he suddenly lost the use of the right arm, and, partially, that of the right leg; articulation was likewise affected.

From this attack, it would seem that he quickly recovered. On the 4th of February (the day preceding that of admittance), he was again suddenly deprived of the use of the right arm and leg.

When I first saw the boy, on the 6th, he was entirely powerless as to the upper and lower limbs on the right side. Sensation was unaffected, as was likewise articulation, but reflex



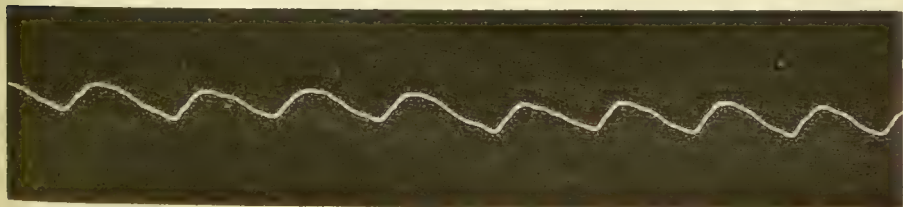
irritability was defective in the paralyzed foot. Pulse 84, and regular. Apex-pulsation in left nipple-line. At the right base a loud *bruit de râpe* was heard with maximum intensity, and from this point it was transmitted through the aorta, quite to the lumbar spine, and into the carotid and subclavian arteries; it was likewise audible, but less distinctly, at the apex. The second sound was remarkably sharp in the aorta, and double during *inspiration only*. To have a teaspoonful of syrup of the phosphate of iron three times daily.

The boy is still (February 24th) under treatment. He has gradually recovered the use of the arm, which he can now lift to his head; but in the use of the leg very little improvement has been effected.

Tested by means of the æsthesiometer, sensibility is undiminished in the paralyzed limbs, whilst the dynamometer registers a pressure equal to twelve and a-half pounds for the paralyzed hand, and a pressure of twenty-seven and a-half pounds for the unparalyzed hand, as reported by my colleague Dr. Nixon. To him I am also indebted for the subjoined tracing of the pulse (Fig. LII.), taken on the 24th February. It is characteristic of obstruction, without regurgitation, at the orifice of the aorta, and shows also some degree of hypertrophy of the left ventricle.

It exhibits shortness and want of verticality in the up-stroke, and roundness of summit.

FIG. LII.



Thomas B.

Aortic obstruction. Pulse 78.

Before I had the advantage of seeing this tracing, I concluded from the position of the apex of the heart, and the force of the impulse, that the left ventricle was hypertrophied. From this I further inferred that the lesion at the aortic ori-

fice was of some years' standing, and had most probably originated in acute endocarditis, engaging the valves, at the date of the attack of scarlatina, three years previously; and, finally, that embolism, by detachment of a flake of fibrin from the aortic valve, and its impaction in one of the arteries of the left cerebral hemisphere, most probably the middle meningeal, was the cause of paralysis on both occasions.

On the first occasion, the embolus was most probably composed of detritus, was quickly disintegrated by the force of the arterial current, and washed away from the obstructed vessel; hence, the rapid recovery which followed. In the second attack, however, there is reason to believe that the plug was of a more compact and solid character, but is now undergoing a slow process of reduction by molecular detachment from the surface.

That the structural lesion causing a murmur of exit is confined to the root of the valve, does not involve its effective portion, and is chiefly of the nature of a deposit from the blood, I judge from the clearness of the second sound, the absence of diastolic murmur, and the harsh and grating quality of the systolic murmur, taken in conjunction with the actual occurrence of embolism.

Hemiplegia from this cause in childhood is of favourable augury, but very liable to recurrence.

The treatment required is of the simplest; but excitement and physical effort, and, indeed, all circumstances whatever which tend to hurry the circulation, should be deprecated.

The absence of muscular contraction or cramps in the paralyzed limbs, and of impairment of sensibility, are worthy of notice, as characteristic of this form of paralysis.

Reduplication of the second sound during *inspiration only*, is not new to me. From the reduplication, I infer that the left ventricle is also dilated. The limitation of this phenomenon to the period of inspiration may, I think, be attributed to a virtual exaggeration of the inequality of capacity existing between the two ventricles at that moment, owing to the facility for disgorgement presented to the right ventricle during the inspiratory effort, whilst no corresponding influence is exercised upon the left ventricle by that act.

CASE LXXXIX.—*Recurrent Rheumatism ; Pericarditis ; Aortic Obstruction and Reflux ; Basic Systolic, and Postdiastolic Murmur ; Transient Systolic Murmur at Apex.*

William G., aged eighteen years, assistant in a wine store, was admitted into hospital, October 25th, 1869, suffering from rheumatic inflammation of the knees, feet, and right wrist, with which he was attacked on the 20th. Was under my care in hospital for a previous attack of rheumatism, complicated with pericarditis, from the early part of the preceding February, to the 17th of March following.

State on second admission. Is remarkably pale. Pulse 72, and scarcely visible. Apex-beat in the nipple line ; a loud, "whiffing," systolic murmur is audible at the apex, and is likewise transmitted to the left axilla and back. At the base, a faint systolic, and a loud postdiastolic murmur are heard. Of these, the former alone is transmitted into the carotid arteries. To have watery extract of opium, gr. j, thrice daily.

November 11th. To continue pill at night only, and take sulphate of quinia, gr. iss, twice daily.

25th. Discharged cured as regards the articular inflammation, but with the cardiac signs unaltered.

Admitted for the third time, April 21st, 1873, with a mild attack of rheumatism of the feet and legs, unaccompanied by swelling. Since date of last report, he has been employed as an engraver on wood. Actual condition : Pulse 96, full, regular, and visible. Precordial dulness extended to the left. Apex beat strong, and perceptible over an area of one inch immediately external to the nipple line. At the apex-point a soft systolic murmur is heard ; this is not audible in the left axilla or back, the second sound being faint, and associated with a feeble distant murmur. In proportion as the stethoscope is slowly shifted upwards and inwards towards midsternum, both these murmurs become gradually more distinct ; at the last mentioned point they attain a maximum loudness, which is maintained, with only slight diminution, through the ascending aorta. The diastolic murmur in both situations is the louder of the two, is soft and blowing, and preceded by a sharp second sound ; whilst

that of systolic rhythm is rough, and alone audible in the arteries of the neck. To have a quinine mixture thrice daily, and gr. j of watery extract of opium at night.

The existence of an indubitable mitral systolic murmur, of temporary duration, at the date of the second admittance, I would venture to attribute either to the accident of mitral thrombosis, or to temporary asthenia of the walls of the left ventricle. I cannot admit *cure* of an actual lesion of the valve, of which I have never seen presumptive evidence, much less proof. During the interval of more than three years between the second and third admittance, disease of the mitral valve, previously established, would have made such progress intrinsically, and would have entailed collaterally such an amount of structural alteration and functional impairment, as would have rendered the diagnosis of it a problem of the simplest character; Whereas, not only had no such changes been effected in the interim, but murmur had ceased to be audible at the apex, except by transmission.

The postdiastolic rhythm of the second murmur suggests to me marginal lesion of the valve only, and that of a trivial character.

The following case affords another example of a murmur of this rhythm, but due to a different and a more formidable lesion.

CASE XC.—*Rheumatism; Dilatation and Atheroma of the Aorta; Harsh Systolic Murmur, accompanied by Fremitus at the Base, and transmitted through the Arch of the Aorta, and into the Arteries of the Neck; also a Soft Postdiastolic Murmur at the Base, and transmitted through the Arch of the Aorta only.*

Michael B., aged fifty-five years, a man of a respectable class, but ruined in circumstances, and desponding, called on me, January 14th, 1870. Had lived rather freely, and nine years previously had rheumatism. Six months prior to the date of his visit to me, he began to suffer from a feeling of discomfort about the heart, with occasional palpitation, and also from vertigo and failure of memory. When I first saw him, he was anæmic; the face was puffed, and of an olive tint. Pulse 96, full, soft, arhyth-



mically irregular, and barely visible. Cardiac impulse very feeble, perceptible half an inch outside the nipple line, and beneath the sixth rib. At the apex, a transmitted systolic murmur was audible. At the base and in the course of the aorta, a very loud systolic bellows-murmur, and a faint postdiastolic murmur associated with a clear second sound, were heard; of these, the former only was transmitted into the carotids. No *arcus senilis*.

February 4th. A loud, harsh, substitutive systolic murmur, accompanied by fremitus also, a soft postdiastolic murmur existed at midsternum; both were transmitted throughout the arch of the aorta, but most distinctly so to the level of the second right costo-sternal articulation. Beyond the left sterno-clavicular articulation the diastolic murmur was not audible; neither was it to be heard in the arteries of the neck; whilst the systolic murmur was heard in this situation with remarkable distinctness. The latter was likewise audible at the apex, but here it was accompanied by a first sound. To have a mixture of iron and chloric ether in camphor water.

I conclude that the aortic valve in this case was extensively disorganized at the root; whilst, as I infer from the existence of a clear second sound, and from the harsh quality of the systolic murmur in the aorta, conjoined with the faltering and irregular character of the pulse, the inadequacy of the valves was due to dilatation of the aorta, involving the orifice.

CASE XCI.—*Palpitation; Sudden and Severe Epigastric Pain, followed by Vomiting and Collapse; Visible Pulsation in the Carotids; Double Impulse; Displacement of the Apex Outwards; Double Basic Murmur Transmitted into Aorta, Systolic Murmur alone being Audible in the Neck; Death. Autopsy: Passive Effusion into the Pericardium; Enlargement of, and Fatty Accumulation upon the Heart; Hypertrophy and Dilatation of the Left Ventricle; Interfascicular Fatty Deposition; Rupture of one of the Segments of the Aortic Valve, and Calcareous Transformation of its Root; Dilatation and Atheroma of the Aorta.*

Anne McE., aged forty-four years, admitted January 26th,

1870. Never had rheumatism. Six months previously, she experienced palpitation for the first time, and on the day prior to date of admittance, was seized with pain in the epigastrium, which was succeeded by vomiting. When admitted, she was still suffering from irritability of stomach, the ejecta consisting of the liquids drunk, mixed with mucus and bile. She was then in a state of collapse, chilled and pallid, the eyes sunken, and the breath cold; the whole aspect being that of a person in the collapse stage of cholera. The pulse was 102, very weak, but regular. Arterial pulsation was visible in the neck, but not in the temples or wrists. Apex-beat half an inch outside the nipple-line, but in the fifth intercostal space; and here a feeble second impulse was felt, and a transmitted systolic murmur was audible.

At midsternum, and in the course of the ascending aorta, a double murmur was heard, that of diastolic rhythm, which was blowing and substitutive, being the louder of the two, whilst the systolic murmur alone was transmitted into the arteries of the neck. To have spirit of chloroform, and hydrocyanic acid draughts.

Died in the course of the same day. About eight ounces of serum were found in the pericardium. This was the product of simple passive effusion, as no trace of pericarditis existed; the serous surface being smooth and transparent. The heart was enlarged, weighing twelve and a-half ounces, and remarkably soft and flabby, expanding by its own proper weight when laid upon the table. There was a good deal of fat on the surface. Examined under the microscope, the muscular fibres exhibited normal striation, and seemed healthy; their diameter varying from 1·776 to 1·332 part of an inch; but between the fasciculi there was a good deal of oil deposit.

The left ventricle was somewhat dilated and thickened. The aortic semilunar valves were healthy at the free borders, and competent to close the axis of the passage; but one of them was ruptured in the centre, the rent admitting the point of the little finger, and permitting free reflux into the ventricle when water was poured into the aorta, whilst the edges of the valves came into close mutual apposition. The root of the ruptured valve

was calcareous, and two rugged masses of chalk-like substance, as large as peas, projected on the ventricular surface of the valve, and must have offered considerable obstruction to the outgoing current. The aorta was dilated above the valves, admitting the points of three fingers, and exhibited atheromatous mottling in an early stage.

The legitimate inference from the history of the patient's last illness and from the actual state of the heart as found after death, is that rupture of the valve-segment took place on the day preceding her demise, and that the shock thereby induced, operating upon an already feeble heart, was the immediate cause of collapse and death.

CASE XCII.—*Rheumatism ; Fright followed by Palpitation, which became Habitual ; Pulmonary Emphysema and Œdema, with Cough, Dyspnœa, and Congestion of Face and Neck ; Visible Pulsation of Superficial Arteries ; Displacement of Apex of Heart Outwards and Downwards ; Substitutive Systolic Murmur at Apex, audible likewise in Left Axilla and Back, with Faint Second Sound ; Double Murmur at Base. Diagnosis : Aortic Obstruction and Reflux, and Mitral Regurgitation.*

James C., aged twenty-eight, a cabman of large stature, and intemperate, admitted February 5th, 1870. Six years previously, had rheumatic pains in legs and arms, for which he was treated in the Hardwicke Hospital for six weeks. Eight years ago, he accidentally drove over a woman, breaking her leg ; was greatly alarmed at the occurrence, and felt his heart palpitate violently. Since that date palpitation has been readily provoked by excitement.

Six months since, his breathing became short, and he began to suffer from cough. Has never had hæmoptysis or œdema. Face and neck somewhat congested. Breathing embarrassed. Pulse 96, regular ; visible pulsation in carotid, temporal, and radial arteries. Cough, with frothy mucus expectoration. There is general emphysema of the lungs, with œdema of the bases of both, posteriorly. Precordial dulness, masked by emphysema of lungs. Apex-beat one inch outside nipple line, and in sixth intercostal space. Impulse diffused, but not strong. At the apex,

a loud, blowing, substitutive systolic murmur existed, audible likewise in the axilla and in the back; second sound faint. At the base there was a double murmur, the diastolic being the louder, blowing, and substitutive. Both were audible in the ascending aorta, and the systolic likewise in the arteries of the neck. Midway between apex and base no murmur was distinctly audible. R Blister to front of chest, dry cupping to back, and a diuretic mixture.

18th. Much relieved as to breathing. Discharged.

Re-admitted on the 21st of March. At this date there was general cedema. No further note of the case has been preserved.

The most noteworthy circumstance in connexion with this case is the coincidence of the first symptoms of cardiac disease with the occurrence of nervous shock. A heart and aorta already in process of tissue deterioration, from the excessive and long continued use of alcoholic stimulants, as no doubt was the case here, and barely capable of performing their respective functions under the most favourable circumstances, cannot, without permanent injury, sustain a sudden revulsion from great emotional excitement or physical effort. Thenceforward, the symptoms of failing circulation will be more and more distinctly manifested; whilst tissue degeneration will make more rapid progress.

Further evidence of the truth of this proposition is supplied by the following case.

CASE XCIII.—*Angina; Severe Mental Shock, followed by Aggravation of Angina, and Œdema of Feet; Pallor and Extreme Weakness; Displacement of Apex-Point outwards; Musical Systolic Murmur at Base, and traceable through Arch of Aorta into Arteries of Neck and downwards to Apex, likewise audible in Left Axilla and Back; Second Sound Clear. Diagnosis: Aortic Obstruction by Calcareous Formation at Root and on Ventricular Aspect of Aortic Valve, Central and Marginal portions being Sound; No Reflux; Left Ventricular Hypertrophy; No Mitral Disease.*

Eliza M., aged fifty years, a ladies' nurse, was admitted into the Mater Misericordiæ Hospital on the 23rd of April, 1870. Up



to two years prior to that date her health had been good, with the exception that, for some time previous, she had occasionally experienced dyspnœa, with pain shooting down the left arm after great exertion. She then received the startling news of the sudden death of her husband; the dyspnœa and pain in the arm became more frequent and more severe, and her feet soon became swollen. She never swooned, and never spat blood.

At the date of admittance, she was pallid. The radial pulse was not registerable, owing to extreme weakness; it was not visible. Feet slightly swollen. No engorgement of liver. No cough. Breathing in the recumbent posture tranquil. Precordial dulness not noticeably extended, but apex-pulsation half an inch external to the nipple-line. Impulse very feeble. At the apex, a loud, musical, substitutive, systolic murmur was heard; it was likewise audible in the left axilla, and at the inferior angle of the left scapula. Here, the second sound was sharp and clear, but weak. At midsternum a musical systolic murmur was likewise to be heard, and of a character and intensity which indicated this as its point of origin. From midsternum it was traceable upwards, in the course of the aorta, to the left clavicle, and also into the great arteries of the neck. It was likewise traceable downwards to the seat of apex-pulsation, losing force gradually, but undergoing no change of quality, as followed in this direction, or in that previously indicated.

At midsternum the second sound was sharp and clear, but faint; it was not accentuated in the pulmonary artery. The interval between the cardiac impulse and murmur on the one hand, and the radial pulse on the other, was not unusually prolonged. To have carbonate of ammonia, wine, and liquid food.

Two circumstances in connexion with this case are worthy of special notice; namely, that a murmur of aortic efflux in the adult was distinctly audible at the apex, in the left axilla, and at the angle of the left scapula; and that advanced disorganization existed at the orifice of the aorta, unaccompanied by inadequacy of the valves.

CASE XCIV.—*Acute Rheumatism ; Basic Diastolic Bellows-Murmur, which subsequently became Harsh ; Displacement of Apex Outwards. Diagnosis : Inadequacy of the Aortic Valves by Roughness of their Edges ; Left Ventricular Hypertrophy.*

John W., aged twelve years, was admitted into hospital, May 12th, 1870, suffering from acute rheumatism of three weeks' duration, and engaging chiefly the joints of the right arm. He was pale, and suffering acute pain. There was visible pulsation of the carotids, but not of the radial or temporal arteries. The apex of the heart pulsated in the nipple line, and here a transmitted diastolic murmur was heard. A loud diastolic bellows-murmur existed at the base, and was transmitted to the apex, but not into the carotid arteries.

Four days subsequently the basic murmur was rasping in quality. Under treatment with colchicum and bromide of potassium, the affected joints being at the same time wrapped in cotton wadding, the boy soon got well of his articular symptoms, and abruptly left hospital without permission.

Judging from the point of apex-pulsation, I am clearly of opinion that the cardiac affection in this case was of, at least, several months' duration, and was probably due to the previous attack of rheumatism, of which, however, no history was obtained.

CASE XCV.—*Exposure and Intemperance ; Œdema, Jaundice, and Hæmoptysis ; Paroxysmal Dyspnœa of an Ascending and Descending Scale ; Pulse Regular and Visible ; Superficial Arteries Tortuous ; Extension of Precordial Dulness ; Apex-Beat in Nipple Line ; Diffused and Heaving Impulse ; both Sounds of Heart Masked at Apex ; Postsystolic-Murmur at Base, and transmitted into the Carotids ; Marked benefit from the use of Chloral ; Repeated Puncturing, and Copious Discharge of Serum ; Gangrene in Seat of Punctures ; Death by Asthenia. Autopsy : Effusion into Pericardium ; Enlargement and Granular Degeneration of Heart ; Hypertrophy with Dilatation of Left Ventricle, and Thinning at the Apex ; Dilatation, Atheroma, and Hyperæmia of the Aorta ; Obstruction at the Aortic Orifice by Thickening of one of the Valve-Segments.*

Laurence C., aged thirty-eight, a gasfitter, and addicted to

tippling, was admitted into the Mater Misericordiæ Hospital, June 2nd, 1870. Has been much exposed to wet and cold in his avocation. Eleven weeks prior to date of admittance, his breathing became short, and three weeks later his feet began to swell.

When admitted he was suffering from orthopnoea, and likewise from paroxysmal dyspnoea, which exhibited the following peculiarities. Whilst he sat propped up in bed, there were alternations of suspended and of quickened respiration, passing into one another by gradations. The apnoeal period, as timed by my watch, lasted twenty seconds, and during its continuance he was in a state of composure, and seemed free from suffering; then, there was a shallow inspiration, which was followed by an expiration of proportionate depth, and this was succeeded by several respiratory acts in increasingly rapid succession, till a rate of one in three seconds was attained, after which the rate gradually subsided to complete apnoea. The ascending scale extended over a period of thirty-six seconds, and in the progressive ratio of two, three, and four respirations in the successive periods of twelve seconds; the descending scale followed in the inverse order, and was of equal length; and finally, the apnoeal period of twenty seconds completed the series.

During the period of ascent, the man moaned and tossed about for breath; he then experienced gradual relief till respiration was entirely suspended, when, as already stated, his tranquillity was complete. These variations of respiration were not attended with any alteration in the rate, force, or rhythm of the heart. Pulse 87, regular, small. Arteries in forearm tortuous, and pulsating visibly. Throbbing of carotids. Great oedema and rigidity of feet, legs, and penis. Conjunctivæ icteric. Urine 1·020 in sp. gr., and albuminous. Precordial dulness much extended; apex-beat diffused, and in nipple line; impulse diffused and heaving, but not strong. Both sounds at apex were somewhat masked. At the base, and in the course of the ascending aorta, and loudest rather above the site of the aortic valves, a soft, prolonged, postsystolic murmur was heard; it was likewise transmitted into the cervical arteries. Second sound not clear, but free from murmur.

6th. Spat some blood this morning for the first time. There has been a copious discharge of serum from punctures made in the penis. He has not slept since admittance, except on the night of the 4th, when he had a draught of grs. xv of chloral hydrate, and slept an hour and a-half. Hoffman's anodyne in 3ss doses failed to give him relief. Night draught of chloral (grs. xx) to be repeated. Chest to be dry-cupped, and one leech to be applied to the precordium.

On the evening of the 13th, he had an epileptiform fit, which lasted about an hour, during which he was unconscious and violently agitated. Subsequently, the thighs became enormously swollen; and from the tension so caused, great pain was experienced at the flexure of the groins. From punctures made in the groins and in the prepuce, there was a profuse discharge of serum.

Citrate of iron and quinine in gr. v doses was given thrice daily, and grs. xx of chloral hydrate at night. Through the agency of the latter he slept soundly every night except two, and on these two nights the chloral had been omitted by an oversight. On the 19th, the swelling of the thighs and legs had begun to subside, and respiration had become tranquil and regular.

28th. Chloral has been continued in similar doses, with the effect of insuring sound and refreshing sleep throughout the night. He usually awakes about 3 o'clock, A.M., remaining awake for about half an hour, and again sleeping till 9.30. Swelling less; breathing tranquil; appetite good. He is very cheerful and hopeful.

In the early part of July the thighs were again punctured; and although he continued free from suffering, and slept well at night under the influence of chloral given in the previous doses, he began to sink. Gangrene set in around the punctures in the thighs, and he died quietly on the evening of July 10th. Permission was granted by the relatives of deceased to examine the heart only. The pericardium contained about eight ounces of amber-coloured serum. The heart was globular, broad at the apex, and weighed twenty-two and a-half ounces. There were two well marked milk spots, one on the anterior, and the other on the posterior



surface of the right ventricle. The former was remarkably opaque, and consisted of hypertrophy of the sub-epicardial connective tissue, by which the serous membrane was sharply raised. The aorta was dilated above the sinuses, admitting three fingers; it was slightly atheromatous, and of a pink red tint on the internal surface. The aortic valves were competent and sound, with the following exception; viz., the right half of the right anterior segment was sharply thickened at the attached border; the thickened portion was smooth on the surface, and projected both on the arterial and ventricular aspects of the valve, less, however, on the ventricular surface, where it was invested by the lining membrane, but still projecting sufficiently to oppose an obstacle to the outflow of blood from the ventricle. The right chambers were normal, except that the anterior surface of the ventricle presented a thin layer of fat. The left ventricle was greatly dilated, thickened to three-fourths of an inch at the base and the middle, but thinned to two lines at the apex. The mitral valves were unaltered. The substance of the heart was somewhat granular. All four chambers contained yellow thrombi; those in the ventricles extended into the aorta and the pulmonary artery. The latter vessel was normal. Both coronary arteries commenced in one of the aortic sinuses.

CASE XCVI.—*Orthopnœa and Cough; Visible Radial Pulse; Weak Cardiac Action; Systolic Murmur at the Apex, and Double Murmur at the Base of the Heart; Systolic Murmur in the Arteries of the Neck; Death. Autopsy: Enlargement of the Heart; Dilated Hypertrophy of the Left Ventricle; Complete Disorganization of the Aortic Valves, and Thickening of the Aorta; Hypertrophy of the Coronary Plexuses.*

Barry W., aged thirty-eight years, a labourer, was admitted, July 23rd, 1870. Had drunk hard, but had never been affected with rheumatism. His breathing had been short for the preceding two years, but he had never spat blood. When admitted, he was remarkably sallow. There was orthopnœa, cough, and mucous expectoration, but no cedema. Pulse 84, soft, and visible. Car-

diac action weak; no impulse to be felt, and no precordial dullness, owing to the existence of pulmonary emphysema.

At the apex point, as localized by auscultation, a soft systolic murmur was heard, and at the xiphoid cartilage, both a coarse systolic and a soft postdiastolic murmur existed; the former was likewise audible in the carotid arteries. To have citrate of iron and quinine, and, at night, grs. xx of chloral hydrate. He became gradually weaker, and died on the 16th of August.

The heart weighed thirteen ounces. There was complete thrombosis of the right auricle, whilst the other chambers contained flakes of fibrin. The left ventricle was in a state of ex-centric hypertrophy. The right and left segments of the aortic valve were rolled up and thickened, and the former was rough and calcareous. The posterior segment was somewhat thickened and shrivelled, but less altered than the other segments. The orifice was reduced to an irregular chink, and was permanently patulous. The root of the aorta was thickened, but not atheromatous. All the other orifices and valves were free from disease. The coronary plexuses were thickened, and the coronary arteries were dilated, but soft and pliant.

*Remarks:* There was a noticeable difference in quality between the systolic murmur at the apex, and that heard at the base of the heart. I cannot offer an explanation of this difference, except by assuming the occurrence of a dynamic murmur at the mitral opening.

The second sound heard to precede the diastolic murmur at the base, was that of the pulmonary artery; the murmur which represented the aortic second sound being posterior in time, owing to the dilated state of the left ventricle and aorta. In this respect, the case illustrates the remarks made at p. 126, in regard to the relative position in time, of the aortic and the pulmonic elements of a reduplicated second sound, in connexion with inadequacy of the aortic valves.\*

\* See also Case 99, last paragraph, p. 887.

CASE XCVII.—*Dyspnœa, Albuminuria, and Œdema; Visible Pulsation of Radial and Ulnar Arteries; Extended Precordial Dulness; Feeble Cardiac Impulse; Systolic Murmur at Base of Heart, and likewise in Ascending Aorta, Arteries of Neck, and at Apex. Diagnosis: Hypertrophy of Left Ventricle; Obstruction at the Aortic Orifice, and Atheroma of the Aorta and Arteries generally; Renal Disease. Marked Improvement; Discharge.*

John F., a waiter, aged forty years, was admitted, November 26th, 1870. Never had rheumatism, and had not been subject to fainting fits or dizziness. During the preceding two years he had noticed that his breathing became short when he was hurried. Three weeks prior to admittance his feet became swollen, then the genitals, and finally the hands. Urine 1·010 in sp. gr., contained albumen, and deposited lithatès. Examined microscopically, it exhibited tube-casts denuded of epithelium, and large crystals of lithic acid. There was œdema of the feet, legs, and external genitals. Pulse 72, regular, and abrupt. Radial and ulnar arteries tortuous, and pulsating visibly on both sides. Breathing tranquil. Precordial dulness extended transversely. No distinct cardiac impulse to be felt, but a sensation of feeble heaving was communicated to the hand over the precordium generally. A harsh systolic murmur existed at the base of the heart and in the ascending aorta; it was heard in the arteria innominata, and likewise, but less distinctly, in the carotids, and at the apex of the heart. The second sound, wherever audible, was sharp and clear. R Tinct. ferri, perchlor. ʒij; Æther. chloric., tinct. nucis vomicæ, a. a. ʒij; Infusi quassia, q. s. ad. ʒviiij. An ounce to be taken thrice daily.

December 9th. Urine again tested. Sp. gr. 1·020, and still containing albumen. Genitals repeatedly punctured since last report. Copious diuresis set in on the 7th, and continued up to the 9th. At latter date, the quantity passed in twenty-four hours amounted to six pints. A decided improvement has taken place in his general condition, and the œdema has nearly disappeared from the lower limbs and genitals.

15th. Left hospital to resume his work. Œdema quite gone, but cardiac and arterial signs as previously reported.

CASE XCVIII.\*—*Intemperance ; Fall upon the Chest ; Dyspnœa and Anasarca ; Visible Pulsation of Superficial Arteries ; Displacement of Apex Outwards ; Double Impulse ; a Systolic and a Prediastolic Murmur at the Right Base ; Sudden Death. Autopsy : Hepatic and Pulmonary Congestion, and Opacity of Capsule of Spleen ; Enlargement of Heart ; Dilated Hypertrophy of Left Ventricle, and Yellow Mottling of its Interior ; Incompetence of Aortic Valve by Relaxation and Perforation of Right Anterior Segment ; Dilatation and Atheroma of the Aorta ; Granular Degeneration of Discoloured Portion of Papillary Muscles.*

— D., a metal founder, aged thirty-four years, and formerly very intemperate, was admitted in September, 1872, under Dr. Nixon, who kindly invited me to examine him.

He had not had rheumatism ; but about two years prior to admittance he fell upon his chest, and sustained injury of an indefinite character, from which he subsequently experienced some slight inconvenience.

Two months prior to the date of admittance his feet became swollen, and his breathing short and oppressed.

At the date of my examination of him, he was very weak. Visible pulsation of radial, carotid, and temporal arteries ; radial pulse feeble, but regular. Apex-pulsation an inch and a-half to the left of the nipple-line. Impulse heaving, rather weak, and double ; the diastolic being the stronger of the two strokes, and accompanied by a faint dull sound. Immediately preceding the diastolic impulse and the accompanying (second) sound, a prolonged bellows-murmur, of medium loudness, was heard at the apex and in the left axilla. The first sound was faint and dull at the apex. At the base, two murmurs were heard ; viz., a faint, dull, systolic murmur, traceable through the aorta and into the neck, and a remarkably loud prediastolic murmur of a blowing character, which was audible throughout the arch of the aorta.

\* This is the case referred to at p. 220, fourth paragraph.



and over the front of the chest generally. It was loudest at mid-sternum, and became gradually less distinct towards the apex. I had commenced my examination at the apex, and my first impression was that this murmur was presystolic, a mistake into which the dull quality of the succeeding sound, and the strong succeeding impulse led me, but which the soft blowing character of the murmur should have enabled me to avoid. On applying the stethoscope over the base of the heart, however, I at once identified the murmur as prediastolic in rhythm. Dr. Nixon was of opinion, in which, however, I could not concur, that a systolic murmur likewise existed at the apex.

The man died quite suddenly a few days afterwards; and on examination of the body, the liver was found to be congested, and the capsule of the spleen thick and opaque. The bases of both lungs were congested. The heart was enlarged, and weighed eighteen and a-half ounces. The right cavities were normal, and contained some clot. The left ventricle was greatly dilated, and its walls were somewhat thickened at all points except the apex, and here the thickness of the wall did not exceed three lines. The interior of the ventricle was somewhat opaque, and mottled yellow. The mitral opening was somewhat dilated, but not disproportionately to the ventricle, and the mitral valve was correspondingly expanded, and competent to close the passage. There was slight thickening of the anterior flap near the edge, but only by extension of the attached and thickened tendinous chord. The left auricle was normal. The aortic valves were atheromatous, especially the right anterior segment, which, when the valve was closed down, hung loosely into the ventricle several lines below the level of the other two segments, leaving a passage as large as the tip of the index finger, by which water poured into the aorta flowed at once into the ventricle. In the centre of the right segment of the valve there was, moreover, an opening as large as a pea, with jagged edges, and near this, another of the diameter of a pin's head. The aorta was dilated, and internally it was somewhat red in patches, and rough by atheromatous change.

Doctor Nixon reports that the discoloured portion of the papillary muscles, examined microscopically, was found to be in the granular stage of fatty degeneration.

CASE XCIX.—*Intemperance ; Strain of Chest ; Œdema, Orthopnœa, and Paroxysmal Dyspnœa ; Visible Pulsation of Superficial Arteries ; Congestion of Liver and Lungs ; Extension of Precordial Dulness ; Strong and Double Impulse of Heart, and Displacement of Apex Downwards, and to the Left ; Systolic Murmur at Apex, and in Left Axilla and Back ; Systolic and Postdiastolic Murmur at Right Base ; Sudden Death. Autopsy : General Enlargement of the Heart ; Dilatation and Thinning of the Right Ventricle ; Dilatation and Thickening of the Left Ventricle ; Slight Thickening of the Mitral Valve ; Softening and Laceration of Two Segments of the Aortic Valve ; Atheroma and Dilatation of the Aorta above the Valves.*

Michael D., aged forty-five years, a brewer's carter, who had drunk freely of porter, was admitted into the Mater Misericordiæ Hospital in October, 1872. Nine weeks previously, whilst engaged with other men in lifting a cask of porter up a ladder, he overbalanced himself, and would have fallen to the ground had he not held on by one of the rungs of the ladder, from which he suspended himself. He felt a strain in the chest for a moment, but soon recovered, and was able to resume his work. Six weeks later his feet began to swell.

When admitted, he was suffering from orthopnœa, with occasional paroxysms of dyspnœa. Respiration 36. Feet and legs much swollen. Pulse 84, regular, full and strong, but visible. Precordial dulness was extended horizontally, and the impulse of the heart was heaving, strong, prolonged, and double. The apex-beat was felt in the sixth intercostal space and nipple line. A soft systolic murmur was audible at the apex, and in the left axilla and back. At midsternum a double murmur was heard ; viz., a faint, somewhat harsh systolic, and a soft postdiastolic murmur, the second sound being sharp and clear. The former of these murmurs alone was audible in the arteries of the neck. Urine 1·015, acid, depositing lithates on cooling, and containing a trace of albumen. Liver enlarged and congested. Lungs congested.

Death took place rather suddenly. On examination of the body, the heart was found enlarged, weighing nineteen ounces,

globular in figure, and exhibiting two large "milk spots" on the right ventricle; viz., one on its anterior, the other on its inferior surface. The pericardium over these spots was smooth and glistening. The right ventricle was dilated and thinned, and both it and the right auricle contained some dark coagulum. The left ventricle was dilated, and its wall at the base and middle was three-fourths of an inch thick. The mitral valve was competent, but slightly thickened at the free margin. The right and left anterior segments of the aortic valve were thickened, brittle, and rent across the centre, the free edges not being engaged, but forming "bridles" across the orifice when the valves were shut down. When the valves were closed, the rents in them readily admitted the point of the little finger. The aorta was atheromatous, and much dilated above the level of the valves.

There is good reason to conclude that the aortic valves, previously to the occurrence of laceration, had been both obstructive and inadequate, and that then, as subsequently, the second sound was double; a murmur of reflux, which was postdiastolic in rhythm, owing to the dilatation of the left ventricle, representing the second sound in the aorta. The second sound of the pulmonary artery was audible at the right base, and preceded the aortic murmur.\* It was, no doubt, owing to the previous existence of regurgitation at the aortic orifice, that accidental laceration of the valves, nine weeks prior to admittance, failed to develop new symptoms of cardiac and respiratory distress. The dependence of paroxysmal dyspnoea on atheromatous change and dilatation of the aorta, and of diastolic impulse upon excentric hypertrophy of the left ventricle, are well exemplified in this case.

CASE C.—*Hæmoptysis; Displacement of the Apex of the Heart Outwards; Double Basic Murmur; Sudden Occurrence of Right Hemiplegia, with Aphasia and Amnesia; Rapid Recovery of the Use of the Arm and Leg, and of the Organs of Speech; Imperfect Recovery of the Remembrance of Words. Diagnosis: Capillary Embolism of the Left Hemisphere of the Brain.*

James M., aged forty years, a corn porter, admitted, November

\* See p. 882, last paragraph.



the 8th, 1872. Never had rheumatism, but spat blood occasionally. Was accustomed to push heavy loads with his arms. Complained of great weakness. Pulse 84, full. Apex-beat in nipple line, and felt over an area of an inch and a-half in diameter, and here two transmitted murmurs were heard. At base two murmurs were likewise audible, of which that of systolic rhythm alone was transmitted into the carotid arteries, and was audible in the left back. The second sound was not intensified in the pulmonary artery.

On the evening of the 20th he was in his usual state, and in bed, having apparently undergone slight improvement in regard to sleep and feeling of weakness. On the following morning he was "queer," and when I saw him at 11 A. M., he answered questions incoherently and with difficulty; was feverish, and the right eye was hyperæmic. I expressed to the class my suspicion that capillary embolism of the brain was taking place.

On the 22nd he was hemiplegic on the right side, scarcely able to utter a word, and quite "astray."

On the 23rd he had, in some degree, recovered the use of the arm and leg, and was more intelligent; the left eye was now more injected than the right.

On the 24th he had recovered the perfect use of his limbs; he answered promptly but irrelevantly, and both eyes were injected.

25th. He was quite intelligent, but had forgotten the proper names of things. When, however, the correct names were mentioned, he at once, and with apparent satisfaction, recognised them, and repeated them without difficulty. When asked the name of an object which he did not remember, he tried hard to recall it, and frowned in the effort to do so. I asked him to name the day of the week (Monday) on which he was admitted; he answered "Thursday," but with apparent doubt as to his accuracy. I then asked him was it not "Monday," and he immediately, and with evident satisfaction, answered, "Yes, Monday." When I placed my finger on his great toe, and asked him what it was, he said "Thursday;" but when I inquired was it not his toe, he answered, with a smile, "Yes, toe."

After a course of treatment with iodide of potassium and



infusion of cinchona, he was discharged on the 3rd December, having quite recovered the use of his limbs and the faculty of speech. His remembrance of the names of things was, however, still imperfect, and the cardiac signs remained unaltered.

CASE CI.—*History of Rheumatism and Severe Pain in Right Side ; Recurrent Angina Pectoris ; Orthopnœa ; Œdema, Cough, and Hæmoptysis ; Feeble Cardiac Impulse and Faint First Sound ; Accompanying Diastolic Murmur at Base ; Death. Autopsy : Effusion into all the Serous Cavities of the Chest ; Heart Enlarged and Fatty on Surface ; Thrombosis of Right Chambers ; Hypertrophy and Dilatation of both Ventricles ; Great Dilatation of Tricuspid Opening ; Valves all Structurally Sound, but Aortic Valves Inadequate by Dilatation of the Aorta, which was likewise Atheromatous ; Osteoid Deposit between Base of Right Lung and Diaphragm.*

John F., aged fifty years, had served twenty-one years in the Metropolitan Police Force; became an extern patient of the Mater Misericordiæ Hospital in the early part of 1866. He was then suffering from dyspnœa, with slight cough and expectoration. I soon lost sight of him; but subsequently learned that in May, 1867, he had an attack of rheumatism, which was accompanied with severe lancinating pain in the lower part of the right side. I next saw him in February, 1868. The feet and legs were then swollen; dyspœna had become much aggravated, and he coughed incessantly.

On the 20th of March following he called at my house in a state of the most extreme respiratory distress, which required for its alleviation the free use of wine and ether, and the admission of air by the window. I now learned that for the previous four weeks he had not been able to lie down, such was the difficulty of breathing in the recumbent posture. He was admitted into hospital on the 21st of March, and at that date his condition was as follows: The feet, legs, and thighs were greatly swollen and tense, but not erythematous. Urine 1·018 in sp. gr., acid, and, on cooling, exhibited a brick-dust deposit which disappeared on the application of heat. There was venous and arterial throb-

bing in the neck, and the supra-clavicular fossæ were obliterated. The area of precordial dulness was much extended, the impulse of the heart was feeble, and over the apex the first sound was somewhat masked and ill pronounced, but free from murmur; whilst the second sound was accompanied by a soft and faint murmur. At midsternum, the first sound was masked and prolonged, but without murmur, whilst the second sound was obscure, and attended with murmur of a soft and blowing character, extending upwards and to the right with scarcely diminished intensity. There was congestion of the bases of both lungs, and respiration was universally feeble, and accompanied with râles. Under treatment, consisting of stimulants and anti-spasmodics, the man improved considerably, so that he was soon able to lie down, and even to sleep in the recumbent posture.

His condition remained nearly the same from the 30th of March to the 4th of April. During this interval, Dr. Grimshaw kindly took the subjoined tracings of the pulse (Figs. LIII. and LIV.) which, as will be seen, are characteristic of hypertrophy with dilatation of the left ventricle, and aortic regurgitation without obstruction. They were in all particulars confirmatory of the diagnosis previously made.

FIG. LIII.

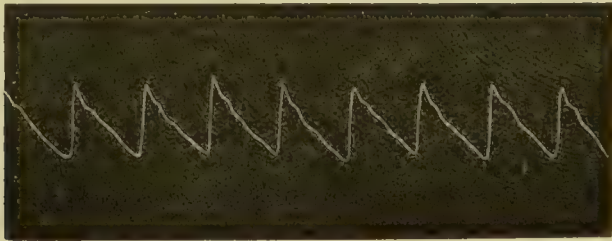
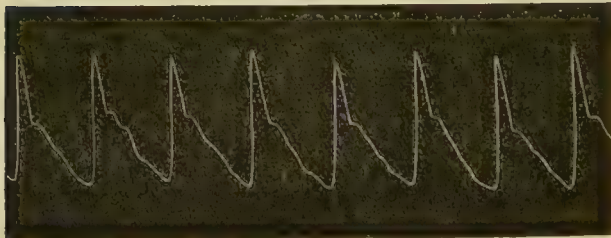


FIG. LIV.



On the 2nd of April there was, for the first time, hæmoptysis;

and this continued without intermission till his death, which took place at 11 P.M. on the 4th of April.

The body was examined fifteen hours after death. There had been very considerable effusion into both pleuræ, and into the pericardium. The left lung was emphysematous, and along the anterior margins of both there had been considerable extravasation of blood. The base of the left lung was likewise, but to a less extent, the seat of extravasation.

The heart was greatly enlarged, fatty on the surface, and weighed twenty-five and a-half ounces. The right chambers were occupied by a large mass of partially decolorized fibrin, which likewise passed for some distance into the pulmonary artery. The right ventricle was somewhat dilated and thickened, and the tricuspid orifice readily admitted the four fingers and thumb. The left auricle had undergone no apparent change, but the left ventricle was greatly thickened and dilated. The mitral orifice was somewhat dilated, but the valves had undergone proportionate expansion, and were healthy and competent. The aortic orifice was greatly dilated, but the aortic valves were healthy, except that the attached margins were somewhat thickened; they were, however, incompetent, owing to the dilatation of the orifice. The aorta, for a distance of three inches above the orifice, was greatly dilated; it was likewise atheromatous, but not rough internally. The lining membrane was hyperæmic.

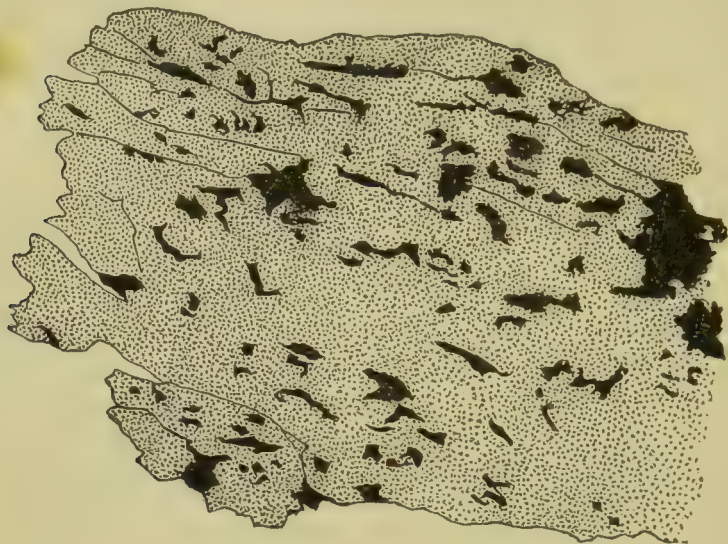
The liver was much enlarged; the spleen was normal; the kidneys were congested, the cortical structure was thickened, and there was a good deal of fat in the pelvis.

Between the base of the right lung and the upper surface of the diaphragm, there was found a plate of osteoid material, two or three lines thick and two and a-half inches in diameter. It was concavo-convex, and strictly adapted to the surface of the lung and the diaphragm, to both of which it was firmly attached. Microscopic examination of this plate showed that it possessed many of the histological characteristics of normal bone. Thus, it exhibited lacunæ, canaliculi, and large channels, not unlike the Haversian canals. They differed, however, in some degree, from the corresponding features of bone, in regard to size, figure, and regularity of arrangement. The annexed woodcut, from an



admirable sketch made with the camera by Dr. E. W. Collins, shows accurately the characteristic features of this adventitious structure.

FIG. LV.



In 1844, Dr. Law exhibited before the Pathological Society of Dublin, a specimen of pseudo-ossific deposit in false membrane attached to the pleura; and he mentioned to me another example which had come under his notice, of a similar deposit between the base of one of the lungs and the diaphragm.

Doctor Hillier brought under the notice of the Pathological Society of London,\* in 1855, a case in which osteoid nodules were dispersed extensively through the soft tissues of the body. He found them in the lungs and in the diaphragm. The illustrations, annexed to his report, of the microscopic characters exhibited by sections of these nodules, resemble Fig. LV. in many respects. Dr. Hillier regarded the morbid formations in his case as cancerous, cancer having existed in other parts of the body. No appearance whatever, even remotely suggestive of cancer, was exhibited in my case.†

*Diseases of the mitral*, as those of the aortic valves, may be

\* *Vide Reports*, vol. vi., page 317.

† For details of the preceding Case (101), see also *Proceedings of the Pathological Society of Dublin*, vol. iii., part iii., new series, page 277.



classified under the two heads of lesions of obstruction, and lesions of regurgitation.

*Obstruction* of the mitral orifice arises, in many instances, from primary inflammation and contraction of the auriculo-ventricular ring, and should be designated annular rather than valvular. In such cases, the disease has its origin in sub-acute inflammation, most frequently rheumatic, of the tendinous ring, engaging the valves secondarily and by extension. It is most frequently met with in children, and is characterized pathologically, in its ultimate stages, by extreme contraction of the orifice, thickening of its boundary, and cohesion and shrinking of the valves, constituting veritable cirrhosis. The aperture thus narrowed, whilst retaining its natural figure, and bounded by seamed, puckered, and well-defined edges, presents on the auricular aspect a very remarkable appearance, appropriately designated the "button-hole" form of contraction. It was exemplified in Cases 102 to 113 inclusive. In adults, and in persons of middle age, more especially those of a gouty diathesis, calcareous nodules are frequently formed upon the edges of the opening by consecutive degradation of tissue. Annular, as distinguished from valvular stenosis, is by many degrees more common at the mitral than at the aortic orifice. This difference depends, no doubt, upon the greater quantity of white fibrous tissue involved in the left auriculo-ventricular ring.

The button-hole contraction of the mitral valve is occasionally so modified as to present the appearance of a diaphragm, stretched horizontally in the plane of the opening, with a central aperture resembling a hole in a drum-head. This aperture frequently retains the elongate figure, and the direction, of the slit exhibited between the closed valves in the normal condition; the edges, however, being thick and tuberculated. On the auricular aspect the outline of the contracted slit is crescentic. The pathology of this modified narrowing is identical with that of the button-hole contraction, which it represents in an early stage. Its origin is usually rheumatic, and the subjects of it are most frequently children or adults.

The form of lesion designated "funnel-shaped" stenosis, is the result of primary acute inflammation and thickening of the valve

segments, with cohesion of their adjacent edges.\* The obstruction thus produced leads to hypertrophy of the left auricle, and from this, in turn, arises increase of pressure upon the valves, which are forced downwards into the ventricle and assume the appearance of a funnel, with a more or less contracted orifice, projecting by its small extremity into that chamber. Cases 102 and 116 furnish examples of this form of contraction.

The relative numerical proportion in which mitral stenosis assumes these two forms respectively, has been variously estimated. Of the 15 cases in which I was enabled to determine this question positively by *post mortem* examination, 13 presented examples of the button-hole form of constriction, and 2 only that known as the funnel-shaped form. Of the former list, 8 were connected with a history of rheumatism; and in 4 of these, aortic was associated with mitral valve-disease. Of the 47 cases recorded by Dr. Hilton Fagge, the contracted mitral opening had assumed the "button-hole" form in 46 instances, and the funnel-shaped configuration in 1 only.† Since my attention was first directed to constriction of the mitral opening, ten years ago, I have had under observation, and have carefully noted, 81 examples of it. Of these, 63 are included in the principal list (Table XI.), upon which my statistics of the disease are based. The remaining 18 cases have been noted since that table was constructed, and are included in the supplementary list. (Table XII.)

The *symptoms* of mitral constriction, disassociated from the physical signs, are by no means characteristic. Most of them may be witnessed in other forms of valvular lesion. A few of these symptoms, however, are eminently suggestive of mitral obstruction, as distinguished from other forms of cardiac disease, and are present at an early period in cases of valvular disease, with rare exceptions, only where this lesion exists. This is especially true in regard to sharp pain occasionally felt at the apex of the heart, and hæmoptysis.‡ It has been stated, and

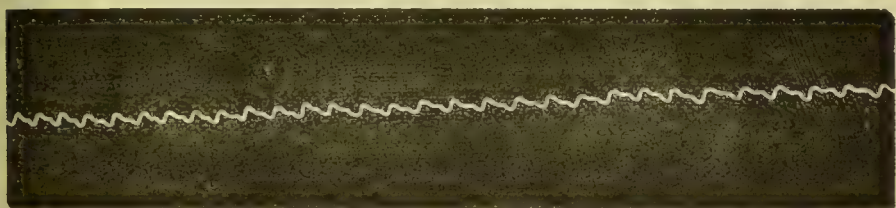
\* See p. 801.

† *Guy's Hospital Reports*, series iii., vol. xii.

‡ I do not contemplate the possibility of confounding this with pseudo- or hysterical pain.

somewhat generally believed, that the pulse of mitral constriction is quick, small, weak, and irregular. In the advanced stages of the disease, when the left ventricle begins to fail, and congestion of the lungs, engorgement of the right chambers of the heart, general venous obstruction and anasarca have taken place, such is, no doubt, the character of the radial pulse (see Fig. LX.); but in these respects it differs not from the pulse of simple mitral regurgitation at a corresponding period of that disease. In the earlier stages, and up to the period when, in the ordinary course of the disease, the changes just mentioned have taken place, the pulse of mitral obstruction is usually quite regular, and not often above 90 in the minute, but small.\* (See Figs. LXII., LXIII., and LXV.) It is, however, easily quickened by exercise or moderate exertion, and is then associated with palpitation, dyspnœa, and pain at the apex. In a few

FIG. LVI.

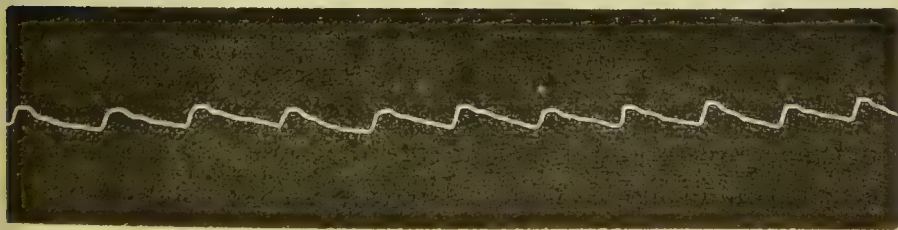


Mitral constriction (extreme).

Michael C. B. Pulse 114. September 22nd, 1873.

I have, unfortunately, lost my notes of this case; it is, therefore, not included in the Tables.

FIG. LVII.



Mitral constriction. Pulse 60.

Thomas D. (No. 39, Table XI.) Slight irregularity and inequality of pulse.  
June 13th, 1873.

\* Dr. Fagge says (*loco citat.*, p. 76), "It has appeared to me that in the great majority of cases in which a presystolic murmur has been heard, the pulse has presented no indications whatever of the existence of disease."



cases the pulse is permanently quick. The annexed tracing (Fig. LVI.) represents the only example of this kind which I have met with.

It may, on the other hand, be permanently slow, as shown in Figs. LVII. and LVIII.

Respiration is habitually short, and readily accelerated by physical effort of any kind, especially by that of making an ascent. There is generally short and teasing cough, with little or no expectoration, and a feeling of tightness in the chest. The temperature is low, the patient is easily chilled, very susceptible to cold, and liable to intercurrent attacks of bronchitis.

Occasional expectoration of pure and florid blood is a common symptom, whilst dropsical effusions are very rare; occurring only in the ultimate stages, when, by failure of the ventricles, pulmonary and general venous congestion has taken place. This remark applies also to congestion of the liver, spleen, and kidneys, accompanied with epigastric tension, and abundant deposit of lithates, with a trace of albumen in the urine. Death is, in most cases, directly caused by oedema of the lungs, which is attended with great difficulty of breathing, orthopnoea, and increased hæmoptysis, and by serous effusion into the pleuræ and pericardium.

FIG. LVIII.



Mitral constriction and regurgitation. Hypertrophy and dilatation of left ventricle. Albuminuria and hæmaturia. Michael O'B. (No. 18, Table XII.) Pulse 36. September, 1874.

The *physical signs* of mitral obstruction are, however, in the highest degree characteristic; indeed, carefully studied and identified, they are pathognomonic of the disease. These signs are two; namely, fremitus and murmur of presystolic rhythm. They have been so fully discussed in a previous chapter,\* that a brief recapitulation of their salient features is alone necessary here.

\* Chapter iii., p. 213.



*The fremitus* of mitral narrowing is located at the apex, and is strictly limited to the apex area. It communicates to the hand a jarring or vibratory sensation; it is usually, but not always, well pronounced; and it immediately precedes the systolic impulse of the heart.

Corvisart fully recognized the value of precordial fremitus as an element in the diagnosis of mitral narrowing. With reference to the signs indicative of this affection, he remarks: "In this number is a particular rustling, difficult to describe, perceptible to the hand when placed on the precordial region, and which, doubtless, proceeds from the difficulty which the blood experiences in passing through an aperture no longer proportionate to the quantity of fluid to which it has to give vent."\*

Apex fremitus, not due to mitral obstruction, is exceedingly rare. It may be caused, however, by mitral regurgitation, the valves being rough or spiculated, as well as inadequate; or by aneurism of the apex of the heart.

The fremitus of mitral reflux, besides being a very rare phenomenon, is strictly systolic in rhythm, and therefore *coincides* with the impulse and the first sound of the heart; whereas, that of mitral obstruction is distinctly presystolic, and *precedes* the impulse, as already stated. Fremitus, due to aneurism of either ventricle, is most frequently *diastolic*, and when not of that rhythm, it is always systolic.†

So characteristic and so easy of recognition is the fremitus of mitral obstruction, that from it alone, on applying my hand to the precordium, and before proceeding further with the examination of the patient, I have repeatedly and correctly diagnosed that lesion. It is not, however, a constant symptom. It is frequently absent where murmur announces, in the most positive manner, that mitral narrowing actually exists. Out of the total of 81 cases included in Tables XI. and XII., it was present only in 41 instances, or a little more than one-half of the whole number; and of the 15 cases in which death occurred, and the body was examined, it was noted in 6 only.

*Presystolic murmur* is, in the strictest sense, pathognomonic

\* *The Diseases of the Heart*, Hebb's translation, p. 206.

† See Cases 69 and 70, pp. 759 and 763.

of this condition. It is *never* present where mitral narrowing does not exist, and it is *rarely* absent, and then only for a very limited period, in cases of that lesion.\* Dr. Hilton Fagge failed to detect it in 4 cases, the patients being at the time *in articulo mortis*. He is, however, of opinion, that "it is yet to be proved that such an affection of the mitral valve can be present in any marked degree, and yet, while the heart is beating steadily and quietly, no presystolic murmur be at any time discoverable."†

In some recent lectures,‡ exhibiting great originality, but promulgating, as I venture to think, very unsound doctrine, Dr. Whyte Barclay has endeavoured to discredit the views generally held by physiologists in regard to the rhythm of ventricular systole, and to modify essentially the current pathology and the significance of presystolic murmur. He alleges that "the sounds of the heart do not indicate either the commencement or the termination of the systole;" that "the ventricles begin to contract before the long silence is concluded;" that "when the ventricle is full, the onward current through the mitral stops, then the systole begins, and before its termination the first sound occurs;" and finally, he urges that it is quite impossible the first sound and the impact on the "ribs can occur, until after contraction has proceeded to some extent." In reference to the last statement, I will only observe that it would imply that the pulsation of the aorta and of the carotid arteries should necessarily precede the first sound; such, however, is not the case.

He surmises that, "owing to some circumstance, the closure of the mitral valves may be postponed, whilst the other elements of the first sound are masked or muffled. Thus, the first sound is sharp, the short pause shortened, and the long pause lengthened, while, during the portion of systole preceding the valve-element, a murmur may occur, which yet may be systolic." But ventricular systole competent to educe valve-click, and of presystolic rhythm, should necessarily be accompanied by presystolic pulsation of the aorta and its primary branches. Hence, carotid pulsation should precede the first sound, which it never does, and presystolic murmur should be synchronous with the

\* See Tables XI. and XII., and p. 901.

† *Loco citat.*, p. 73.

‡ *Lancet*, March 2nd and 16th, 1872.

carotid pulse, which it is not. He recognizes two distinct stages of ventricular systole; namely, "tension," and "shortening" of the fibres, "the tension of the fibres being complete when the impulse is felt, and the first sound occurring somewhere in the middle of the contraction, at the point, in fact, where tension is complete and shortening begins." Finally, he regards presystolic murmur as due to mitral regurgitation during the state of "tension" of the ventricle.

But ventricular tension, adequate to the development of a reflux murmur at the mitral orifice, should likewise cause efflux by the normal outlet, and pulsation of the carotid arteries; whilst its maximum force, exerted "at the middle of contraction," and causing the impulse and the first sound, should, in every such case, give rise to a second and louder murmur of systolic rhythm. Now, it is scarcely necessary to urge, that the true presystolic murmur does *not* coincide with carotid pulsation, but *precedes* it by a distinctly appreciable interval; and that mitral systolic murmur is rather an exceptional than an ordinary accompaniment of presystolic murmur.

Doctor Cryan has brought under the notice of the Pathological Society of Dublin, an example of mitral and tricuspid narrowing, in which presystolic murmur could not be detected, although the patient had been fifteen days under his observation in hospital, and during that time had been repeatedly examined by him.\* From the general features of the case, notwithstanding the absence of presystolic murmur, the diagnosis of mitral narrowing was made. Dr. Cryan thinks that the absence of murmur may have been due to extreme contraction of the mitral orifice, which was, in this case, actually reduced to the diameter of the point of the little finger; the tricuspid opening being likewise greatly contracted. The diagnosis was very creditable to Dr. Cryan's acumen; but his explanation of the failure of murmur is to me not entirely satisfactory. I have had under observation examples of still greater narrowing of the mitral opening, in which presystolic murmur was well pronounced, and continued to be heard throughout.

\* *Proceedings of the Pathological Society of Dublin*, vol. iv., part ii., new series, 1870

Doctor O'Ferrall was of opinion that, in many instances, narrowing of the mitral orifice is consecutive to, and compensatory of, shortening of the mitral valves from previous endocarditis.\* He adduces, in support of this opinion, three cases which came under his notice. In these cases there was systolic murmur, with fremitus, at the apex; the murmur ceased sometime before death, and the mitral orifice was found greatly contracted. He believed that, in endocarditis, the valves are affected before the orifice; hence regurgitation precedes obstruction. In proportion as the orifice is involved at a later stage, it is narrowed, and so adapted to the shortened valves; hence the cessation of a previously existing murmur. This ingenious theory is based upon a fact and a postulate. The segments of the mitral valve were found to overlap one another; hence "the mechanism of the parts seemed incompatible with regurgitation;" and a murmur of mitral constriction, if present, "should have been in connexion with the second sound." It is most probable regurgitation had not at any time existed in these cases; that the murmur actually present in the first instance was presystolic, a supposition certainly not excluded by the test of rhythm applied; and, that its suppression at a later period was due to the extreme debility which is admitted to have been present.

Doctor W. R. Sanders has found the presystolic murmur to be that which has varied most remarkably. He adds, that exertion on the part of the patient is generally, but not always, competent to educe it, where mitral narrowing exists.†

Doctor W. R. Gowers, on the other hand, maintains that the assumption of the erect posture has precisely the opposite effect upon this murmur. Drawing his conclusion from his experience of four cases, he asserts that the presystolic murmur is "louder and longer in the recumbent than in the erect posture," and that "in many instances it may be heard in the former, and not in the latter posture."‡ In reference to the effect of change of posture, my experience rather coincides with that of Dr. Sanders. In no instance have I found the murmur of mitral constriction less

\* *Dublin Journal of Medical Science*, vol. xxxii., July, 1843.

† *Edinburgh Medical Journal*, vol. xiv., 1868.

‡ *Practitioner*, December, 1873.



loud in the erect, than in the sitting or recumbent posture. Indeed, in every case of the kind that has come under my notice the murmur in question has been intensified by the effort of standing up.

Doctor Waters believes that presystolic murmur is "far more frequently absent than present, even when there is great obstruction at the mitral orifice."\* He gives five examples within his own experience in support of this opinion. The first of these patients was under observation nearly a month; the second, for several weeks; the third, a few hours; the fourth, two months, and subsequently for five days; and the fifth, nearly three weeks. In all these cases, mitral systolic murmur was heard, but presystolic murmur not at any time. The mitral orifice was contracted in each of these cases.

Where extreme debility from any cause exists, the murmur is most frequently suppressed in the agony of dissolution, and this may continue for several days.† It may be in abeyance for a longer or shorter period, according to circumstances. Thus, I have repeatedly failed to detect it on the first examination of a patient recently admitted, and still suffering from exhaustion due to want of proper food, and to the fatigue attendant upon removal to hospital. Even in such cases, however, suspicion as to the nature of the case having been aroused by the preceding and attendant symptoms, an ill developed murmur may, by strained attention, be detected in the presystole. From this, in conjunction with other evidence, I have ventured, in several instances, to make the presumptive diagnosis of mitral obstruction, and have found my diagnosis confirmed by well pronounced murmur on the following day. In 2 cases only, out of the 66 not fatal, has a presystolic murmur, previously identified, ceased to be audible whilst the patient was under observation. After two days, however, it returned in 1 of these cases, and continued to be heard during several weeks, till she was discharged. Three years subsequently, this patient was again received into hospital, with the signs of mitral narrowing still more distinctly pronounced. In the second case, the cessation of murmur coincided with great

\* *Diseases of the Chest*, second edition, 1873.

† See Case 108.

weakness and irregularity of pulse, and was again audible on the following day.\*

Of the 15 fatal cases in which mitral contraction was proved by autopsy to have existed, there were only 3 in which presystolic murmur was not at any time heard. In one of these cases the patient was nine days under observation previous to her death. She was, however, hemiplegic and aphasic, and the heart acted with extreme feebleness and irregularity during the whole of that period.†

The second case was under observation five days, and, although in a very weak state, the patient was not moribund during the greater portion of that time. I examined her daily, and I am convinced presystolic murmur cannot have then existed. Its absence, as already stated (p. 712), I am disposed to attribute to enfeeblement of the left auricle, by the pressure of a copious effusion into the pericardium and pleuræ.‡

The third case afforded the most remarkable example of suppressed presystolic murmur which has come under my notice. I use the word "suppressed" advisedly, because, in view of the condition of the mitral orifice discovered after death, I concluded that murmur of this rhythm must, of necessity, have existed at an earlier period of the man's illness. He had been thirty-nine days under observation, had been repeatedly examined, and the *seat* of the murmur heard had been frequently discussed with my colleague, Dr. Nixon, and with the class; but its *rhythm*, which was by all of us regarded as systolic, had never been called in question. I am satisfied, therefore, that presystolic murmur had not been pronounced at any time during the man's residence in hospital. I can offer no explanation of the suppression in this, as in the cases previously referred to, except the debility of the heart.§

In Case 68 (p. 749) presystolic murmur was inaudible when the patient was first examined, neither was it heard for several days after my second visit to her. It was then detected, and

\* See Cases Nos. 8 and 9 in Table XII.

† See Case 108.

‡ See Case 61, p. 710.

§ See Case 114.

continued to be distinctly audible from that date till a short time before her death. The body was not examined.\*

In the case of the woman, Jane McG. (109), presystolic murmur was not detected till eighteen days after her admittance. During this period the pulse was very weak, and the heart was repeatedly examined. For some time preceding death, the action of the heart was not equable, and murmur was heard only during the strong contractions of the ventricle.†

Thus, in 2 cases only out of a total of 81, or in the proportion of 2·4 per cent. was the murmur actually suspended under my personal observation, yet in both of these it was resumed after a brief interval. In the first, I cannot explain the suspension; but in the second, great weakness of the heart existed.

Amongst the fatal cases, presystolic murmur was not at any time heard in 3 instances out of 15, or in the proportion of 20 per cent.; and in a third case, it was detected only after the patient had been eighteen days under observation. In a fourth case, not included in this list, because the body was not examined after death, murmur was detected only a week after the patient had come under my notice.

In the case of the girl Eliza B. (No. 12 in Table XII.), presystolic murmur was developed during a secondary attack of rheumatism. I conclude that in this case the mitral orifice had been previously narrowed, but not in a degree adequate to the production of murmur.

In every instance in which I have heard a veritable presystolic murmur, I have confidently diagnosed narrowing of the mitral or tricuspid orifice, according to the seat of the murmur; and of both orifices, where the locality of murmur was dual. Yet, in no single instance has the diagnosis so made been falsified by the event.

Judging strictly within the limits of my own experience, and drawing my conclusions from the statistics above given, I feel justified, therefore, in holding,

- (a) That presystolic murmur is pathognomonic of auriculo-ventricular narrowing;

\* See Case No. 14 in Table XII.

† See Case 109, and No. 7 in Table XI.

- (b) That it is never present where auriculo-ventricular narrowing does not exist ;
- (c) That it is never permanently absent in cases of this lesion ;
- (d) That it is very seldom temporarily absent in such cases ;
- (e) That in these exceptional instances, the explanation of its absence is almost invariably to be found in debility of the heart ;
- (f) That the form of cardiac debility with which the suspension of presystolic murmur is most frequently associated, is that of the process of dissolution ;
- (g) And, finally, that the duration of suspension depends upon that of cardiac debility, and *in articulo mortis* it may extend over several days ;

Presystolic murmur may exhibit four different varieties of rhythm, as already stated.\* I have, in more than one instance, observed these several varieties to shade off into one another, in the order in which they are set down, concurrently with the progress of the disease. The second form has been discussed at p. 206, and also in connexion with an illustrative case at p. 697.† The third form is exemplified in Case 117.‡ The prolonged murmur may undergo an ulterior change of abbreviation, by suppression of its terminal and intermediate portions, the postdiastolic portion alone remaining, as exemplified in Case 116. Even an alternate fusion and resolution of the postdiastolic and presystolic elements of this compound murmur may be observed to take place, in periods of alternating excitement and compression of the heart. Case 117, affords an example of this kind. The fourth form indicates a rough state of the constricted mitral orifice, and great debility of the left auricle ; a condition, in short, proximate to that with which extinction of murmur is associated. It is exemplified in Case 113.

Doctor George Balfour is virtually in agreement with me in

\* See p. 214.

† The cases mentioned at p. 214 as illustrative of this variety of the murmur indicative of mitral narrowing, are Case 116 in the text, and Nos. 39 and 48 in Table XI.

‡ See also Nos. 15, 39, and 42, Table XI.



reference to the variation of form three, or rather he has anticipated me in the view just stated.\* He says, in effect, that the murmur (presystolic) often follows the second sound immediately, being separated by an interval from the presystole, but sometimes running through to the apex-beat. These two forms are, in his opinion, indicative of great contraction of the orifice. The constriction, as he truly remarks, is usually of the diaphragmatic kind; but I doubt he is equally correct in holding that prolonged murmur and sharp first sound indicate a funnel-shaped condition of the mitral opening.

Doctor Whyte Barclay has recorded a case in which a protracted diastolic pause was entirely covered by a double murmur; the first portion faint, soft, and commencing immediately after the second sound; the second loud, harsh, and ending in the first sound.† This was, in my view, an example of the modified form of presystolic murmur which I have described in category number two.‡

Taken alone, and in its typical form, the presystolic murmur of mitral stenosis may be represented by the series *r-r-r*; and in combination with the first sound, which is frequently clear and abrupt, it may be mimicked phonetically by the formula *r-r-r-up*, sharp. The postdiastolic element, whether alone or combined, is, however, usually of soft and blowing quality. Indeed, I can recall only one case (116) in which it was harsh. It presented this character for a brief period only, and was then accompanied with fremitus. Presystolic murmur is strictly confined to the area of the apex in the great majority of cases; and when audible beyond this boundary, it is so only to a very limited extent, very faintly, and manifestly by transmission. I have heard it in the back in one case only (Case 106); in the axilla, in three cases (Cases 106, 117, and No. 12, Table XI.); and I have seldom heard it more than two inches to the right of the point of apex-pulsation.

Fauvel, as already stated (p. 196), was the first observer who fully identified the rhythmical peculiarities of presystolic murmur at the apex. His memoir was published in 1843. Nine-

\* *Edinburgh Medical Journal*, November, 1871.

† *Lancet*, March 2nd, 1872.

‡ See p. 214.

teen years earlier, however, Bertin recognized it, and actually founded thereon the positive diagnosis of left auriculo-ventricular contraction.\* In one case (*XLIX<sup>e</sup> Observation*), he describes the murmur as diastolic in rhythm, accompanying the contraction of the auricles (which, in his estimation, coincided with the second sound), and as attended with *frémissement*. This was, no doubt, an example of the prolonged (third) form of murmur, modified by suppression of its terminal portion as just explained, indicating extreme contraction of the mitral orifice. Its placenary quality may be referred to as confirmatory of this view.

In a second case (*LI<sup>e</sup> Observation*), he describes the murmur as "preceding the pulsations of the ventricles;" thus indicating that its rhythm was presystolic. In both these cases the mitral orifice was found, on *post mortem* examination, to be contracted in an extreme degree.

That Hope, fifteen years after the publication of Bertin's book, failed to identify the murmur indicative of mitral obstruction, is sufficiently manifest from his having described it as "attending the ventricular diastole and second sound;" as "always very feeble, soft, like the bellows-sound, and usually on a rather lower key than a whispered *who*;" and as "exceedingly rare." He adds: "I have never known purring tremor accompany a diastolic (*i.e.*, presystolic) mitral murmur, the current being too feeble to produce it."† He would account for the circumstance that Laennec had regarded this murmur as by no means rare, by assuming that he had mistaken for it the murmur of aortic regurgitation. I incline to believe that the preceding reference to his own writings is not unlikely to raise a suspicion in the minds of many, that he has himself fallen into the error which he imputed to Laennec.

The typical presystolic murmur may be simulated more or less closely, as already stated,‡ by

- (a) Pericardial friction-sound of single and systolic rhythm, confined to the area of the apex;
- (b) Prolonged systolic murmur replacing the first sound at the

\* *Traité des Maladies du Cœur*, 1824, pp. 176 and 186.

† *Diseases of the Heart*, third edition, 1839, p. 387.

‡ See p. 217.

apex, and extending over the short pause; the second sound being associated with impulse;

(c) Reduplicated first sound audible at the apex.

Since the above was written, I have been enabled to supplement the list of possible sources of error by the addition of,

(d) Postdiastolic murmur of aortic origin transmitted to the apex; and

(e) Prediastolic basic murmur transmitted to the apex; the second sound being accompanied by impulse.

Cases 4 and 6 afford good examples of pseudo-presystolic murmur arising from pericardial friction. The differential diagnosis of the pseudo-murmurs (a), (b), and (c), has been already discussed;\* that of (d) and (e) may be now briefly pointed out. The former is well exemplified in the following case. A young man of nervous temperament, and the subject of repeated attacks of rheumatism, was received into hospital on the 21st of August, 1874. The radial pulse was slightly visible, the impulse of the heart strong, and the apex-pulsation felt in the nipple-line. A faint murmur of postdiastolic rhythm was heard at the apex, which corresponded to that described at p. 904, as an abbreviation of the third form of presystolic murmur. At the right base, however, and in the ascending aorta, the second sound was sharp, and was succeeded by a loud bellows-murmur which was traceable to the apex, where it presented the characters already mentioned. The murmur was thus shown to be aortic, not mitral, in origin. Case 89 affords an example of the same kind, but more complicated. Prediastolic basic murmur, audible at the apex by transmission, may be mistaken for presystolic murmur, if, in the latter situation, the second sound be accompanied by impulse; thus assuming the association, and apparently the rhythm, of the first sound. The existence, however, of prediastolic murmur at the base, traceable to the apex, with progressively decreasing intensity, will readily establish the distinction. Case 98 exemplifies these remarks.

Although presystolic murmur, as previously stated, is pathognomonic of mitral stenosis, it is liable to be mistaken for more

\* See pp. 217 and 218.

ordinary phenomena, and even when identified, it is often misinterpreted.

Doctor Alexander Silver, of the Charing Cross Hospital, in a thesis of great merit,\* remarks that owing to the altered character of the first sound at the apex in mitral constriction, and to its close resemblance to the second sound, which is not here audible, whilst the presystolic murmur covers the normal long pause, the latter is often mistaken for a murmur of systolic rhythm replacing the first sound, the real first sound being mistaken for the second.

I have no doubt this mistake has been, and is still, frequently committed in cases exhibiting the combination of signs mentioned by Dr. Silver; but the misleading concurrence is by no means so common as he has been led to conclude from the observation of the case, so fully and so graphically reported in his monograph. In the greater number of examples of mitral stenosis, the long pause is *not* entirely covered by the presystolic murmur; the second sound is *not* always inaudible at the apex; nor does the first sound resemble the normal second sound in all cases.†

Doctor Silver would regard a sharp first sound, unaccompanied by systolic murmur, as indicative of the "funnel-shaped" form of narrowing; the peculiar character of the sound being, in his opinion, due to the collision of the valve-segments "by surfaces, not by edges." Dr. Whyte Barclay would, on the other hand, interpret a prolonged presystolic murmur in the same sense.‡ I do not think the particular form of narrowing actually present can be affirmed, either from the character or accompaniments of the first sound, or from the length of the murmur. Cases 104 and 111 show conclusively that a sharp and clear first sound at the apex, unaccompanied by murmur, may be associated with mitral narrowing of the button-hole character.

Finally, in reference to differential diagnosis, it is necessary to observe, that when mitral inadequacy coexists with mitral obstruction, the murmurs indicative of the twofold lesion run into

\* Prize Essay, *Medical Times and Gazette*, March 16th and 23rd, 1873.

† See Cases 102 to 116, inclusive.

‡ *Loco citat.*



one another, constituting a single prolonged murmur. This may be, and no doubt in many instances has been, regarded as entirely systolic. But the harsh character of the presystolic, contrasted with the usually soft and blowing quality of the systolic element, and the coincidence of impulse and of carotid pulsation with the latter, will render the distinction not difficult.\*

Professor Purser thinks it "possible that a powerful auricle, a dilated ventricle, and an orifice (mitral) of normal size, might be as effective in producing a murmur (presystolic) as a ventricle of normal size and a narrowed orifice. In each case the due relation between the size of the orifice and the size of the ventricle being disturbed in the same direction."† But such, in fact, is not the case. No example has been recorded and verified by dissection, of presystolic murmur arising from dilatation of the left ventricle, or occurring in the absence of positive mitral narrowing. Dilatation of the left ventricle, from incompetency of the aortic valves, is of very common occurrence, yet, presystolic murmur is not associated, even exceptionally, with that condition. Theoretically, I would say, that the deranged relationship which determines presystolic murmur, is not that between the mitral orifice and the left ventricle, but that between the volume of blood descending from the left auricle and the force with which it is propelled, upon the one hand, and the diameter of the mitral opening, upon the other. I may add that, as a matter of fact, I have never satisfied myself of the existence of presystolic murmur, where, on *post mortem* examination, I have not found the mitral opening absolutely narrowed.

Amongst the morbid changes directly due to obstruction at the mitral orifice, sanguineous engorgement and extravasation in the lungs is one of the most common. Hope remarks that he had met with pulmonary apoplexy more frequently in connexion with great contraction of the mitral orifice, with or without hypertrophy of the right ventricle, than in any other association.‡ Intercurrent expectoration of pure florid blood is rarely absent in the course of the disease. It is of frequent occurrence

\* See Cases 102, 110, and 113.

† *Irish Hospital Gazette*, March 15th, 1873.

‡ *Opus citat.*, p. 249.

in the greater number of examples; and, in the early stages of the disease, as a means of relieving the temporary congestion of the lungs, and as being unlikely, at this period, to be attended with extravasation into the pulmonary structure, it need not be deprecated;\* neither should it be the occasion of alarm to the patient. The quantity discharged is usually moderate, and in no instance have I known it to be the cause of sudden death. When hæmoptysis occurs, however, within the few last days of the patient's life, it may be regarded as an infallible indication of pulmonary apoplexy; it is then accompanied by great respiratory distress, general venous engorgement, and dropsy. Hæmoptysis has occurred in 44 out of my 81 cases of mitral obstruction; and of the 15 fatal cases, pulmonary apoplexy was found to have existed in 8.

The term "apoplexy," as applied to these extravasations in the tissue of the lungs, is not only awkward, but inappropriate. That of "hæmorrhagic infarction," first applied by Virchow,† is much preferable. According to him, the condition so designated is one of extreme capillary engorgement and extravasation into the vesicular and interstitial structure of separate lobules of the lung, consequent upon embolism of the branch of supply from the pulmonary artery to these lobules. The vessels beyond the plug being shut off from the pressure of the right ventricle, tension within them is lowered, and there is necessarily an afflux to them from neighbouring capillaries by collateral pressure. The blood so entering the lobule, being no longer under the direct influence of the heart, cannot be expelled. Stasis and capillary hæmorrhage are the result, coagulation extending into the veins.

Lefevre holds that there is simple engorgement, by paralysis, of the capillary walls;‡ whilst Feltz, on the other hand, maintains that infarctus is characterized by actual extravasation.§

Ludwig, as quoted by Niemeyer,|| considers that an explanation may be found in simple stasis, and precipitation of the blood-

\* See Case 104.

† Traube's *Beitrage zur Experimentellen Pathologie, u Physiologie*, Berlin, 1846, and Froriep's *N. Notizen*, Nr. 794.

‡ *Etude Physiologique et Pathologique sur les Infarctus Viscéraux*, Paris, 1867.

§ *Traité Clinique et Expérimentale des Embolies Capillaires*, Strassburg, 1870.

|| *A Text-Book of Practical Medicine*, 1869, vol. i., p. 156.

corpuscles within the capillaries, consequent upon the reduction of vascular pressure arising from contraction of the main artery. But, on this hypothesis, the actual occurrence of extravasation by which pulmonary apoplexy is distinguished, cannot be accounted for. The objection to Virchow's theory, that it is based upon an assumption which anatomy does not sanction, namely, that of an anastomosis between the pulmonary plexuses of adjacent lobules, seems fatal to it. It is, however, possible that a collateral circulation, adequate to the production of capillary engorgement and hæmorrhage within the lobule, may be indirectly established through the bronchial system of vessels, which Dr. Waters has shown to be in direct communication with the pulmonary veins.\*

This view as to the pathology of sanguineous infarction of the lungs will satisfactorily account for the multiple, circumscribed, and nodular form in which it is presented; whereas, these characteristics could not be explained under any theory implying general and equal vascular pressure in the lungs, whether *a tergo* in the pulmonary artery, from an hypertrophied right ventricle, or *a fronte* in the pulmonary veins, from obstruction or reflux at the mitral or the aortic orifice.

Assuming, as I do, with the qualification above mentioned, that Virchow's doctrine is correct, I should expect to find hæmorrhagic infarction of the lungs most frequently, if not invariably, accompanied by thrombosis of the right side of the heart. Clots have been actually found in the right chambers in 11 out of the 15 foregoing fatal cases of mitral stenosis; and in 8 of the former, pulmonary apoplexy likewise existed. Hence, hæmorrhagic infarction of the lungs was, in every instance, accompanied by thrombosis of the right chambers of the heart. The subjoined case, which has come under my notice whilst the preceding paragraphs were being written, lends considerable support to the doctrine of Virchow. Indeed, it seems to me to establish that doctrine conclusively. The case is given in abstract, as follows:

Agnes F., a child nine years old, was admitted into hospital on the 10th of August, 1874, under my colleague, Dr.

\* *The Human Lung*, 1860, p. 201.



Nixon. She was suffering from renal anasarca following scarlatina. A few days after admittance, she suddenly became hemiplegic on the left side. The paralysis was exclusively motor, and only partial in degree. She quickly recovered the use of the affected limbs; but the anasarca continued, despite active treatment. The urine was loaded with albumen, and exhibited epithelial tube-casts. About the middle of August I took charge of the girl, in the absence of Dr. Nixon, and for nearly a month subsequently she continued under my observation. During that time she had an attack of pleuritis with effusion on the right side, from which she made a good recovery. She continued, however, weak and pallid; the feet were slightly swollen, albumen was still present in the urine, and the pupils were persistently dilated. Her condition fluctuated, even from day to day, between extreme debility with partial coma, and a bright and cheerful manner, accompanied with perfect intelligence. Dr. Nixon resumed charge of her about the 6th of September; she was then moribund, and on the 10th she died comatose. The body was carefully examined by Dr. Nixon, to whom I am indebted for the report of the *post mortem* examination, as well as for the favour of obtaining the subjoined engraving. There was considerable effusion into the right pleural cavity, and likewise into the pericardium. The heart was structurally normal, but the right chambers were occupied by a decolorized and friable thrombus. The superior lobe of the left lung exhibited, in its antero-inferior portion, a mass of hæmorrhagic infarction as large as the last joint of the thumb. It was dark, well defined, and perfectly solid; and on section it presented the appearance of black-currant jelly. The two branches of the tertiary division of the pulmonary artery leading to this mass, which included several lobules of the lung, were full, to distension, of decolorized and disintegrated fibrin similar to that found in the right ventricle. The primary division of the pulmonary artery, the tertiary branch leading to the infarcted lobules, and all the adjacent branches of that vessel, as well as those of the pulmonary vein, were empty and collapsed, whilst the lung-structure was elsewhere normal. The condition just described is well represented in the woodcut. (Fig. LIX.)



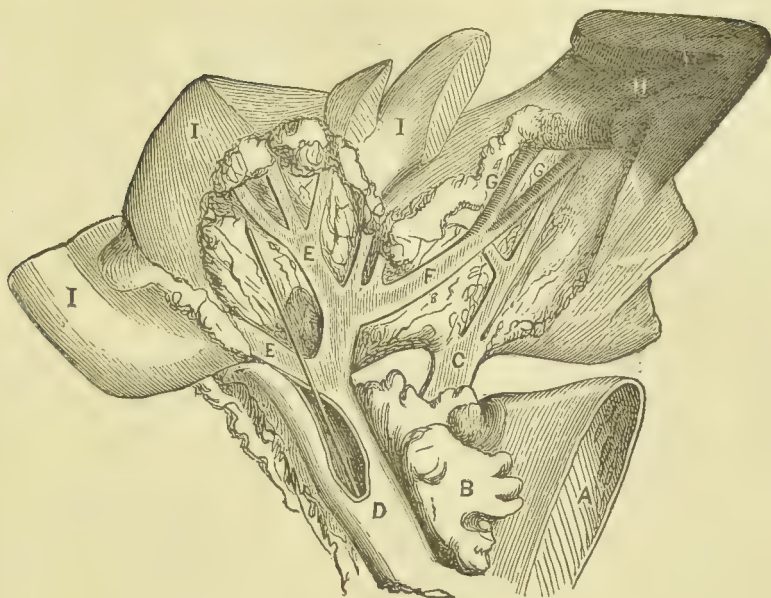


FIG. LIX.

Hæmorrhagic infarction in left lung. (Agnes F.)

- A. Section of left auricle.
- B. Appendix of left auricle, distended by a thrombus.
- C. Left superior pulmonary vein quite empty.
- D. Superior branch of primary division of left pulmonary artery. A bristle is represented as introduced into an opening made in the vessel.
- E. Secondary branches of pulmonary artery empty and collapsed.
- F. Tertiary branch leading to infarcted vessels.
- G. Quaternary and infarcted branches of pulmonary artery.
- H. Infarcted lobules.
- I. Healthy lung-substance.

Dropsy, as previously stated, is of exceptional occurrence in the course of the disease, preceding the final illness; it is then, however, all but invariably amongst the lethal symptoms, and is general. Gangrene of the lower limbs I have not witnessed in more than one instance (Case 114). In this latter respect, disease of the mitral valves, of whatever kind, contrasts strikingly with valvular lesion at the orifice of the aorta. This difference is, no doubt, due to the frequent association of general arterial disease with the latter affection.

With regard to the chambers of the heart, the left auricle is

invariably dilated and hypertrophied, its lining membrane thick and opaque, and the orifices of the pulmonary veins much dilated. I have not witnessed aneurism of the left auricle, of which Thurnam has collected nine examples, in connexion with mitral narrowing.\* The left ventricle, on the contrary, is always normal as to capacity and thickness, where mitral inadequacy or either form of aortic valve-lesion is not likewise present, and no general cause of hypertrophy or dilatation exists. Hope was certainly in error in stating that "the left ventricle is sometimes rendered hypertrophied by a contraction of the mitral orifice."†

Doctor Adams, who wrote on this subject three years before the first edition of Hope's book appeared,‡ more correctly stated that in cases of mitral contraction, whilst the left auricle is dilated and thickened, and the pulmonary veins and right chambers are likewise dilated, the left ventricle is rather diminished in capacity.

In no instance which has fallen under my notice has hypertrophy or dilatation of the left ventricle been fairly attributable to mitral obstruction. In the progress of the disease the right ventricle is invariably dilated, and sometimes hypertrophied, whilst the right auricle is dilated and thinned.

The *treatment* of obstruction at the mitral orifice resolves itself into that of the symptoms and sequelæ of the affection. In this respect, however, much may be accomplished by well directed measures, even in the advanced stages of the disease, towards the alleviation of suffering and abatement of urgent symptoms. The mechanical impediment to the circulation once established, is not removable by medicine. The heart is, however, in an eminent degree capable of adapting itself to the altered circumstances under which it is required to function, when, as is usually the case in regard to mitral narrowing, the progress of the disease is slow and regular. The object of treatment at this period should be, therefore, to favour the process of self-adjustment in the heart, by the avoidance of over exertion, and of mental excitement.

If the heart should exhibit evidence of weakness by intermit-

\* *Medico-Chirurgical Transactions*, vol. xxi.

† *Diseases of the Heart*, p. 250.

‡ *Dublin Hospital Reports*, vol. iv., 1827.

tent or irregular action, quick and feeble pulse, or by threatened syncope, it should be promptly strengthened by the administration of digitalis, either alone or in combination with iron.

Whilst on this subject, I cannot avoid quoting the pertinent and philosophic remarks of Dr. Foster, of Birmingham. In reference to the compensating power of the heart, he writes: "The propelling power must be augmented, to drive the blood at a greater speed, or the time for its passage must be lengthened.

"When the narrowing is not very great, and the demands on the circulation by exercise are limited, a very perfect compensation is effected by the hypertrophy and dilatation of the left auricle and the right ventricle. By these means the current of blood through the lungs is made more rapid, the tension in the auricle is increased, and the velocity of the current filling the left ventricle is so multiplied, that the balance of the circulation is fairly maintained, in some cases for years. But when the narrowing is considerable, or some unusual strain has disturbed the unstable equilibrium on which the health of these patients depends, then the compensation which the auricle and ventricle offer fails. The rapid irregular pulse tells of the varying quantity on which the ventricle contracts. Sometimes the interval between the ventricular contractions is so great, and the ventricular charge is so small, that the systolic wave does not reach the wrist. Under these circumstances the second indication must be followed; the auricle must have more time to fill the ventricle. This, to say nothing of the increased power given to the cardiac muscle, is exactly what digitalis effects. By slowing the action of the heart, the period of time during which the blood from the distended auricle can flow into the ventricle is increased, and as the extra time allows more blood to pass through the narrowed mitral orifice before the final effort of the auricle is made, that effort is made on a smaller quantity of blood, and is, consequently, more effective; for the smaller the quantity of blood which the auricular muscle has to push before it, the greater will be the velocity given to the current. The ventricle, though contracting less frequently, contracts more effectually. Instead of eighty or ninety irregular contractions per minute, no two succeeding ones equal in force, and some so

valueless that they are not perceptible at the wrist, we get some sixty steady equal beats; the pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognizes its master, and beats a sullen retreat."\*

I usually prescribe ℥xv of the tincture of digitalis, with an equal quantity of tincture of the perchloride or acetate of iron, and ℥x of spirit of chloroform, to be taken in an ounce of water every third hour. If pain exist near the apex, or to the inner side of the nipple, I regard it as evidence of debility, tension, and parietal congestion of the right ventricle or left auricle, as the case may be, and proceed on the same principle to exhibit digitalis and iron, or quinine and strychnine, grs. ij of the former with ℥iij of the Pharmacopœial solution of the latter, every third or fourth hour. At the same time, I direct one or two leeches to be applied at the seat of pain, and subsequently an opiate plaster.

Moderate and healthful exercise should be enjoined, in order to promote nutrition, and prevent hepatic and portal congestion. It should not, however, be carried to the point of fatigue, or excitement of the circulation. The food should be selected with special reference to its nutritive and digestible qualities, and no less with a view to the avoidance of flatulence which, by making upward pressure upon the heart and lungs, may give rise to palpitation and dyspnœa. The bowels should be kept free by means of aloetic and saline aperients; and alcohol, except in very small quantity, and by preference in the form of good claret, should be forbidden. Malt liquors are especially liable to disagree with the patient, owing to their tendency to cause portal congestion. Copious imbibition of liquids is to be prohibited, because the volume of the blood is thereby, for the time, increased. For this reason, condiments which provoke thirst must be avoided.

Hæmoptysis, in moderate quantity, need not cause alarm. The condition which it indicates, namely, congestion of the lungs,

\* *British and Foreign Medico-Chirurgical Review*, No. xc., July, 1871; and *Clinical Medicine*, 1874, p. 101.



and engorgement of the right side of the heart, is best treated by active purgation, dry-cupping the chest, and, if the discharge be copious or persistent, by the use of ergot or ergotin. ℞xx-xxx of the liquid extract of ergot may be given in half an ounce of cold water every second hour, till the hæmorrhage cease, or gr. ss of ergotin in solution may be injected hypodermically at corresponding intervals, till the object is attained. The latter is decidedly the more efficacious method of employing this agent.

The treatment of dropsy, so common in the advanced stages of the disease, should be directed, in the first instance, to the relief of congestion of the kidneys, which are functionally impaired by sanguineous engorgement. Six to eight ounces of blood may be taken from the loins by cupping or leeching; or, if the patient be very weak, dry-cupping alone should be practised. In either case a succession of poultices to the loins, as warm as can be borne, should be maintained. The bowels should, at the same time, be well moved by means of compound powder of jalap, grs. xxx, with grs. ij of calomel, in the form of electuary; or, ʒij of the acid tartrate of potash dissolved in a wineglassful of the infusion of senna, with ʒj of the tincture. It may be necessary to give sedatives in order to induce sleep; for this purpose, grs. xx of chloral hydrate, or grs. xv of this, with ℞x of liquor of the hydrochlorate of morphia, may be given in an ounce of water at bed-time.

If the nature of the case be early recognized, and the patient judiciously and continuously treated as here recommended, a protracted period of moderately good health, and even of social enjoyment, may be confidently guaranteed. The patient should, however, be warned that indiscretion of any kind, especially such as may induce acute congestion of the lungs, or otherwise overtax the power of the heart, is likely to be followed by serious consequences.

Amongst the succeeding cases are many which, thus treated, have continued for several years, little if at all, aggravated by lapse of time.

In fine, it may be averred that this is, *par excellence*, the form of organic disease of the heart which is most amenable to early

and appropriate treatment, so far as refers to grave consequences and premature death.

The following 16 cases will be found to illustrate the preceding remarks, and to present, in epitome, the history and pathology of mitral stenosis. They have been all under observation and treatment for some time, most of them for a protracted period; and, finally, a careful *post mortem* examination of the body was made in every instance.

The cases which were not fatal, 66 in number, are presented in summary in Tables XI. and XII., and are explained, where necessary, by marginal notes. The fatal cases are likewise included in these Tables, with a view to obtaining complete results under the several heads mentioned.

CASE CII.—*No History of Rheumatism; Orthopnœa and Palpitation; Œdema; Quick and Faltering Pulse; Strong Impulse; Hæmoptysis; Pain at Precordium and Extending down Left Arm; Angina; Rough First Sound with Presystolic Murmur at Apex; Soft Systolic Murmur at Base; Reduplication of Second Sound; Subsequent Development of Systolic Murmur at Apex; Suppression of Presystolic Murmur for One Day, and of Radial Pulse during the Last Week of Life, with very Slow Respiration; Coma; Death. Autopsy: Effusion into Pericardium; Dilatation and Thrombosis of Right Chambers of Heart, and Dilatation of Tricuspid Orifice; Dilatation and Hypertrophy of Left Auricle; Great Contraction and Funnel-shaped Transformation of Mitral Orifice; Emphysema and Sanguineous Infarction of the Lungs.*

Anne K., aged twenty-eight years, a seamstress, was admitted into hospital on the 24th of November, 1865. Never had rheumatism. Has repeatedly spat blood. Three years previously, she complained, for the first time, of palpitation and breathlessness after unwonted exertion of any kind. Two years later, she had cough; and for four months preceding admittance, she had been unable to lie down, owing to the difficulty of breathing which the attempt to do so occasioned. Her feet had been swollen for the last month.

The face was somewhat congested, and the eyelids puffed, but there was no lividity of the lips or extremities. Pulse rapid, extremely weak and faltering; lower extremities œdematous. The cervical veins were enlarged, and distended, and the external jugular pulsated synchronously with ventricular systole, but the lightest pressure above the clavicle promptly arrested this pulsation. The chest was abnormally resonant, and permanently distended, as in the state of inspiration; the impulse of the heart was distinct and vigorous.

The apex pulsated at a point rather internal to the usual position. At this point the first sound was rough; it was immediately preceded by a harsh murmur which extended up to, but did not implicate it, and was loudest at the apex. At midsternum, a soft, localized, and accompanying systolic murmur existed. The second sound was clear, but reduplicated at midsternum.

The following diagnosis was made: Mitral stenosis, hypertrophy of left auricle, dilatation of both right chambers and of right auriculo-ventricular opening, pulmonary emphysema.

On the 17th of December there was hæmoptysis, the blood discharged being florid, and mixed with frothy mucus; and on the 31st the patient complained of acute pain located in the region of the heart, and extending down the left arm. This pain was anginal in character, being accompanied by a sense of constriction in the chest, and by great respiratory distress.

About the middle of January, a well pronounced systolic murmur was developed at the apex. It was continuous with the presystolic murmur, constituting an appendix to it, but distinguishable from it by its less harsh quality, and by its rhythm. On the following day the presystolic murmur was suppressed; it was not again heard, whilst that of systolic rhythm continued audible up to the patient's death.

The radial pulse was entirely suppressed during the last week of the patient's life; still she continued to move about, and declared she felt better. Two days before her death, which took place on the 26th of January, she expectorated a large quantity of dark blood; respiration, at the same time, fell to 15 in the minute, at which rate it continued to the end. She died comatose.

The *post mortem* examination revealed the following condition of organs: Both lungs were emphysematous, and in the apex of each there was an insulated and well defined mass of very dark effused blood. These portions of the lungs were indurated and non-crepitant. The bases of the lungs were slate-coloured, and contained some extravasated blood; and on the surface of both there were several emphysematous projections filled with solidified blood. The pericardium contained six ounces of serum.

The heart, especially on the right side, was greatly distended with dark clotted blood; it had lost its natural figure, and become greatly expanded transversely. The right ventricle was considerably dilated, and its walls were slightly thickened. The right auriculo-ventricular opening was very large; but its valves, though incompetent, were healthy, as were likewise those of the pulmonary artery. The left auricle was large, its walls were much thickened, and the orifices of the pulmonary veins were slightly dilated. The left ventricle was rather below the normal size; its walls were not thickened. The mitral orifice was much reduced in diameter. Both segments of the mitral valve were thickened and united in such a manner as to form a funnel-shaped passage about an inch and a-quarter long, and projecting into the left ventricle; the opening at the ventricular end of this passage barely admitted the point of the little finger.

A loose and pendulous flake of fibrin was attached to the auricular surface of one of the valve-segments, in such a position that, during life, it must have vibrated in the influx-current from auricle to ventricle.\* The aorta and its valves were perfectly sound.

\* See *Proceedings of the Pathological Society of Dublin*, vol. ii., new series, 1865, p. 179. I do not now hold the opinion then expressed, that the vibration of this shred of fibrin was the cause of the presystolic murmur mentioned in the report of the case.



CASE CIII.—*Rheumatism ; Intemperance ; Mechanical Injury in Region of Liver ; Hæmorrhoids ; Dyspnœa and Lividity ; Rapid and Feeble Pulse ; Anasarca ; Ascites, and Pleural Effusion ; Legs Punctured ; Diminished Secretion of Urine ; Hæmoptysis ; Gangrene of Feet ; Death. Autopsy : Ascites ; Liver Contracted, and in "Nutmeg" Condition ; Spleen Thickened in its Capsule, and containing Masses of Mortar-like Substance ; Bases of Lungs Congested ; Effusion into Pleuræ and Pericardium ; Thrombosis of Right Auricle ; only two Valve-Segments at Right Auriculo-Ventricular Orifice ; Hypertrophy of Left Auricle ; Great Contraction, and Button-hole Figure of Mitral Opening ; Aortic Valves Slightly Inadequate.*

Jane G., aged thirty-four years, a native of Liverpool, and of intemperate habits, was received into hospital on the 26th of January, 1866. Whilst crossing over to Dublin, six weeks previously, she was exposed to wet and cold on the deck of a steam-boat, and on her arrival her breathing was much embarrassed, and her feet and legs became swollen. The poor woman informed me that at the age of thirteen she had an attack of rheumatic fever, from which, however, she suffered no permanent impairment of health ; she occasionally suffered from bleeding hæmorrhoids. Three years prior to admittance, she received from her husband, whilst he was drunk, a blow with the clenched hand in the region of the liver. She was knocked down by the blow, and suffered great pain in the abdomen for some time afterwards. Somewhat later she had ascites and anasarca, and for the latter her legs were punctured. After recovery from this attack she felt her respiration permanently embarrassed.

State on admittance : Face, especially lips, quite livid, and conjunctivæ injected with dark blood ; extremities cold and livid, and feet and legs highly cedematous and tense ; several minute openings had formed spontaneously on the legs, most probably in the seat of former punctures, from which serum freely exuded. Pulse rapid and very feeble, but regular. No visible pulsation of arteries to be anywhere detected. Urine reduced to three ounces in twenty-four hours, and coloured with

bile pigment. There was partial effusion into both pleural cavities.

The impulse of the heart was strong, and attended with *frémissement*. The apex pulsated in the normal position, and at this point two murmurs were audible; viz., a loud and harsh presystolic murmur, which was immediately succeeded by a clear first sound, accompanied by a soft and blowing diastolic murmur. The former was confined to the area of the apex, whilst the latter was derived from the base, where it was loudest, and manifestly of aortic origin; it was traceable upwards in the course of the aorta for a distance of about two inches.

The liver projected considerably below the costal cartilages; it was firm and even on the surface. There was considerable effusion of serum into the peritoneum. The patient's fingers were distorted, and the joints enlarged, apparently from chronic gout.

The legs were again punctured, but they yielded very little serum; and on the following day patches of incipient gangrene appeared upon the dorsum of either foot. The patient was now pulseless. In the course of the succeeding night she coughed a good deal of dark blood, and died at 1.30 A.M.

The body was examined thirteen hours after death, with the following result: The liver was contracted, and in the "nutmeg" condition; its investments, both serous and fibrous, were much thickened. The spleen was of normal size; its capsule was opaque and greatly thickened, and in its parenchyma two large masses of a yellow putty-like substance were found, which seemed to consist of fat and urate of soda; kidneys healthy. There was a considerable effusion of serum into both pleural cavities. The bases of both lungs were solid, and the investing pleuræ were dense and opaque. The pericardium contained a pint of straw-coloured serum, in which a large shred of false membrane floated loosely, having no attachment to either surface. The heart appeared as if daubed over with white paint; it was, however, smooth and glistening on the surface, and of average size.

The right auricle contained a large thrombus, which extended to the auriculo-ventricular opening, through which it projected

by a round bulbous extremity; it did not, however, extend further. The right ventricle was scarcely altered, but the auriculo-ventricular valve was *bicuspid*. The left auricle was much thickened and dilated. The left ventricle was rather reduced in capacity, but thickened. The mitral orifice was of a button-hole figure, and greatly reduced in size, scarcely admitting the tip of the little finger. The mitral valve was thick, rigid, and all but completely calcified; it projected into the ventricle, but the segments lay evenly together so as to close the opening. The aortic valves were thick and rigid; they admitted of slow leakage into the ventricle when water was poured into the aorta above them. The lining membrane of the ascending portion of the arch of the aorta was red; it was likewise dotted with yellow patches of atheroma in an early stage of formation.

Measurements: Right auricle normal; right ventricle, walls  $\frac{3}{16}$  of an inch thick at apex, and  $\frac{3}{8}$  at middle; left auricle, walls  $\frac{1}{4}$  of an inch thick in sinus; cavity, measured from septum to outer wall,  $2\frac{1}{4}$  inches, from superior wall to mitral valve,  $2\frac{3}{4}$  inches, from anterior to posterior wall, 3 inches; left ventricle, walls  $\frac{5}{16}$  of an inch thick at apex,  $\frac{1}{2}$  an inch at middle, and  $\frac{5}{8}$  of an inch at base; cavity, measured from root of mitral valve to apex,  $2\frac{1}{2}$  inches, from anterior to posterior wall,  $1\frac{1}{4}$  inch; ascending aorta, 1 inch in diameter.\*

CASE. CIV.—*History of Scarlatina; Cough, and Repeated Hæmoptysis; Dyspnœa; Weak and Failing Pulse; Anasarca and Ascites; Apex-point Displaced Outwards; Presystolic and Systolic Murmur; Sounds of Heart Sharp and Clear; Sudden Death. Autopsy: General and Firm Adhesion of Pericardium; Slight Dilatation and Thickening of Right Ventricle; Dilatation of Tricuspid Opening; Great Dilatation and Hypertrophy of Left Auricle; Extreme Contraction of Mitral Orifice; Left Ventricle Normal; Substance of Heart Healthy; Aorta of Small Calibre, and Pink Internally; Valves all Healthy, except Mitral.*

Thomas N., aged ten years, admitted February 3rd, 1868.

\* See *Proceedings of the Pathological Society of Dublin*, new series, vol. iii., part i., p. 22.

Five years previously, he had scarlatina, and spat blood. When admitted, he had cough with mucous expectoration; respiration was quick, and the pulse was scarcely perceptible. He spat blood about once every three weeks, the hæmoptysis continuing three days, and being always attended with considerable alleviation of his suffering. The most urgent symptoms were weakness, and shooting pain in the region of the heart; and the chief physical sign was presystolic murmur at the apex, which pulsated immediately inside the nipple-line. He remained under treatment till the 9th, when he was discharged at his own request, much relieved, but without any change of the apex-murmur. The patient presented himself at the hospital on the 17th of February, and again, on the 23rd of March.

On the latter occasion the following note was made: Radial pulse not perceptible; cardiac pulsations 120 in the minute; respirations 36. There has been no hæmoptysis for the last few days; but it may be brought on at once by exertion, such as fiddling with carpenter's tools, to which he is much addicted. Cough continues through the night, and almost entirely prevents sleep. Appetite failing, and stomach irritable; chest prominent, and supra-clavicular fossæ obliterated. Both a presystolic and a systolic murmur are now audible at the apex, which pulsates in the nipple-line. To have a sinapism to chest, and an anodyne expectorant. He continued to spit blood in large quantity, and was re-admitted on the 22nd of June. He was then in a state of unconsciousness; his face puffed and pallid, lips livid, and no pulse perceptible at the wrists.

He was universally anasarcaous; the abdomen was distended with liquid, and there was strong and heaving pulsation of the carotids on both sides. Respiration was accompanied with coarse crepitus. Precordial dulness was extended, and the impulse of the heart was prolonged and heaving. At the apex, a faint but rough presystolic murmur was heard; it was followed by a sharp clicking first sound. The second sound was heard at the apex after every second or third pulsation only; it was clear, but less sharp than the first sound. To have, thrice daily, a draught containing ℥x of chloric ether, spirit of nitrous ether, and aromatic spirit of ammonia respectively; wine, and light nourishment.



25th. He died suddenly at 1 o'clock to-day. The body was examined the following morning. The pericardium was universally and firmly united to the heart by old adhesion. The right ventricle was thickened and dilated, and the tricuspid orifice enlarged. The left auricle, especially the appendix, was greatly dilated and thickened. The left ventricle was small, and normal as to thickness of walls; its substance was firm.

The mitral orifice was reduced to the size of the barrel of a goose-quill; the valves were adherent, except in the centre, where an oval opening of the above mentioned size existed; the edges of this opening were seamed and regular.

The aorta was small, and its lining membrane was of a pink colour which was not removable by washing; the aortic and pulmonary valves were healthy and competent.

CASE CV.—*Fright after Child-birth, followed by Jaundice; Hæmoptysis; Presystolic Fremitus and Murmur; Reduplication of both Sounds of Heart; Intercurrent Pericarditis; Typhus Fever; Death. Autopsy: Hydro-pericarditis, and Roughening of Surface of Heart by Recently Effused Lymph; Enlargement of Heart; Dilatation of Tricuspid Opening; Dilatation and Thickening of Left Auricle; Great Narrowing of Mitral Orifice, and Thickening of Valves and of Tendinous Chords; Liver Olive-Coloured and Fatty; Biliary Ducts Dilated, but not Obstructed; Scirrhus of Pancreas.*

Maria F., aged thirty years, admitted June 18th, 1868; has had one child, which is five months' old. One month after confinement she was frightened by the abrupt entrance of a strange man into her room whilst she was in bed. Four days after this occurrence her friends noticed that she was jaundiced. The jaundice was not attended with pain, nor was it preceded by illness of any kind. She is now deeply jaundiced, of a dark olive colour; urine deeply tinged with bile pigment, and not abundant. Nothing noticeable in region of liver, except some slight tenderness in scrobiculis cordis. At the apex of the heart a soft systolic murmur is audible; at the left base both sounds are double, but no murmur is to be heard here.

June 19th. Urine loaded with bile, but less deeply coloured than formerly. Harley's test for bile acids gave a dark opaque stratum, not purple. Has been taking blue pill, taraxacum, and dried soda; also nitro-muriatic acid in infusion of chiretta.

July 1st. Pulse 96, of good volume, and regular. For the last week there has been less of the colouring matter of bile in the urine; complains of a "fluttering" at pit of stomach. On making a careful examination of the heart to-day, I discovered a very distinct and characteristic presystolic murmur at the apex. It is somewhat rough, and faintly audible for a short distance to the right; both sounds are double, but not very distinctly so at the base; impulse of heart in normal situation.

11th. Admits she spat a little blood, but only once, about three weeks prior to admittance. She experiences a sensation in the skin as if "mill-dust were sprinkled over her"; last night she had a sharp stinging pain in the region of the heart.

On July 27th the patient was attacked with acute pain in the head, accompanied with general febrile symptoms. There was tumultuous action of the heart, and a single, loud, grating sound at the apex, which masked the previous murmur. She was at once put under mercurial treatment; a leech was applied to the precordium, and followed by a succession of warm poultices.

August 3rd. Her condition was as follows: viz., Pulse 108, with occasional intermissions for the first time. No increase in extent of precordial dulness. At apex, a distinct *frottement* was perceptible by the hand, and here a loud jarring and superficial murmur was heard, synchronous with the second sound, but likewise covering the greater part of the long pause; it masked effectually the endocardial murmur previously heard. At a short distance to the right a presystolic murmur was faintly heard, manifestly by transmission. No friction or other abnormal sound heard in this situation, both cardiac sounds being here normal. The grating sound was transmitted into axilla from apex, but with progressively decreasing distinctness.

I left town on the evening of August the 3rd, and did not again see the patient till the 11th. She had, in the *interim*, contracted typhus fever of a most virulent character. The maculæ were large and dark, and interspersed with petechiæ. When I

visited her she was comatose; pulse rapid and feeble. She died in the afternoon, and on the following day (the 12th) the body was examined.

The pericardium contained nearly a pint of serum. The heart weighed eleven and a-half ounces. The inferior surface of the right ventricle was somewhat rough and granulated. The right chambers were normal, but the tricuspid orifice was greatly dilated, admitting four fingers introduced edgewise. The left auricle was dilated and thickened. The mitral orifice was surrounded by a dense white cartilaginous rim, formed by the altered valves; it was circular in figure, and admitted only the tip of the little finger. The left tendinous chords were thickened, shortened, and very rigid. The left ventricle was normal. The liver was of the ordinary size, of a deep olive colour, soft, and lardy, and easily broken down with the finger; the gall bladder was contracted and empty. The hepatic, cystic, and common biliary ducts were greatly dilated; the latter readily admitted the end of the thumb, but the entrance into the duodenum was not obstructed. The head of the pancreas was enlarged and hardened; it was dense and rigid on section.\*

CASE CVI.—*Mechanical Injury of the Chest; Subsequent and Repeated Attacks of Dyspnœa, Palpitation and Collapse; Intercurrent Congestion of the Lungs, and Hæmoptysis; Presystolic, and Subsequently Systolic Murmur at the Apex of the Heart; Soft Systolic Murmur at the Xiphoid Cartilage; Variola; Pericarditis; Thrombosis of the Heart and Sudden Death. Autopsy: Congestion of the Lungs; Partial Pericarditis; Enlargement of the Heart by Hypertrophy and Dilatation of the Right Ventricle; Thrombosis of the Right Chambers; Hypertrophy and Dilatation of the Left Auricle; Great Contraction of the Mitral Orifice; Left Ventricle Normal.*

Robert McB., aged fourteen years, and healthy looking, was received into hospital on the 3rd of September, 1867. He had

\* For this and the preceding case, see *Proceedings of the Pathological Society of Dublin*, new series, vol. iv., part i., p. 18.

enjoyed good health; had not had rheumatism, nor had he been otherwise seriously ill up to a year previous to date of admittance, when he was thrown from a pony in the street, and the animal trod upon his chest. This accident was followed by great pain in the chest; also by dyspnoea and expectoration of florid blood.

After a short respite, he resumed his duties as light porter in a grocer's shop, and for the succeeding eight months enjoyed comparatively good health. He was then suddenly attacked with dyspnoea and palpitation of so severe a character that he was obliged to sit down in the street. Seizures of a similar kind occurred from time to time; and, in one of unusual severity, which occurred on the day of admittance, he fell down in the street, and was carried to the hospital in a state of collapse.

When admitted, he was cold and pallid, the lips and extremities were livid, and respiration was rapid and oppressed; the heart pulsated tumultuously; the radial pulse was 150, and very weak.

On the following morning (4th) I examined him for the first time. His condition was then as follows: Pulse 84, and quite regular. Respiration 24. He was still pale, but quite conscious; the lips were livid; the chest was resonant throughout, and respiration was everywhere normal. The external jugular veins pulsated synchronously with ventricular systole. The impulse of the heart was strong; the apex pulsated in the normal position, and with ordinary force. At the apex-point a presystolic murmur existed, of so typical a character as to warrant, on the instant, the diagnosis of mitral narrowing. Both sounds of the heart were distinctly pronounced, and quite clear. At the base of the xiphoid cartilage a loud bellows-murmur of systolic rhythm was heard; it was traceable for two inches upwards in the course of the aorta. Beneath the spine of the left scapula the presystolic murmur, and also both sounds of the heart, were heard. The second sound was slightly intensified in the pulmonary artery. His condition improved so much that he was discharged at the end of a month, free from all urgent symptoms. The treatment consisted in the application of two leeches to the pre-



cordium, and the administration of infusion of the American wild cherry,  $\text{ʒviiss}$ , with  $\text{ʒij}$  of chloric ether and of aromatic spirit of ammonia, respectively; a tablespoonful to be taken thrice daily. In the event of a paroxysm of dyspnœa occurring, a draught was to be given, consisting of  $\text{ʒss}$  of compound spirit of ether, and  $\text{℥x}$  of chlorodyne, in  $\text{ʒj}$  of camphor water.

He had no return of paroxysmal dyspnœa after the first evening of his sojourn in hospital; but he spat blood repeatedly. On one occasion his pulse fell to 60. Before his discharge, the murmur at the ensiform cartilage had ceased to be audible.

He was again admitted on the 27th of November, under Dr. Hughes. In the interim, he had repeatedly spat blood.

He next presented himself on the 1st of May, 1868. Hæmoptysis then occurred whenever he walked quickly; he could lie only on the right side; the second sound was double at base, otherwise the physical signs were as last noted.

During the remainder of the year 1868 he was re-admitted several times for acute congestion of the lungs with bronchitis. In these attacks he suffered from great dyspnœa, and spat blood repeatedly; but when relieved, as he usually was, by a short course of treatment, including tincture of the perchloride of iron with digitalis, he was able to resume his work as a porter.

The intervals between his admissions varied from weeks to months, the longest having been from May, 1868, to August, 1869. During these intervals he occasionally reported himself at the hospital, but enjoyed moderately good health, and was able to perform his duties, which were sometimes laborious.

When he came under my care in August, 1869, the apex of the heart was displaced to the left, and occupied a position in the nipple-line. A presystolic murmur, more harsh than previously, existed at the apex. A systolic murmur was likewise audible in the same situation. After a short course of treatment, his health improved, and he left hospital to resume his work.

He was subsequently admitted on the 2nd of October, 1869, on the 29th of June, 1870, and on the 12th of May, 1871. On these several occasions he was suffering from acute pulmonary congestion, with hæmoptysis, from which, as previously, he ob-

tained prompt and complete relief under rest and ordinary treatment.

He was admitted for the last time in the early part of November, 1871. His most urgent symptoms then were cough and dyspnœa. Whilst in hospital he had two attacks of angina pectoris, which were of the most aggravated character and nearly fatal; he was then suffering from dyspnœa, which was associated with congestion of the lungs, and bronchitis. The apex of the heart pulsated rather feebly half an inch outside the nipple-line, and in the sixth intercostal space. The presystolic murmur was now masked by a loud systolic murmur, which was diffused over the entire front of the chest, but was not transmitted into the carotids.

His general health was much improved, and he was about to be discharged on the 4th of December, when, unfortunately, he contracted smallpox, some cases of which were then in the hospital. For two days he progressed favourably, having been successfully vaccinated in childhood, and his arm presenting two well-formed scars. The eruption was discrete, and attended with very slight fever; it was out on the third day, and on the fifth day the pustules were full. In short, the case, as one of variola, was in every respect of the mildest character. On the evening of the 9th (fifth of the disease), he was suddenly attacked with dyspnœa and faintness; and, when I visited him on the following morning, he was very weak and breathed with great difficulty. The face was congested, the cervical veins turgid, respiration of a gasping character, and the pulse small, but regular; the cardiac phenomena were as last noted.

Examination of the chest afforded no satisfactory explanation of the unfavourable change which had taken place; the lungs were slightly congested at the base, but respiration was everywhere audible, and expectoration was free, and of the character usual in chronic catarrh.

He continued in this state till the 15th, propped up in bed, moaning and gasping for breath. On the morning of that day he died suddenly, and without a struggle, after getting into bed from the night chair.

The body was examined twelve hours after death. The lungs

were voluminous and congested, especially at the base, but no extravasation had anywhere occurred; both apices were attached to the chest-wall by old adhesions; a few cicatrices existed in the left apex, and the internal surface of that lung was firmly attached to the pericardium. The sac of the pericardium contained no liquid, but its lining membrane was rough on the right side from recent effusion of lymph, and the corresponding portion of the surface of the right auricle exhibited a similar condition. I believe this was the result of pericarditis developed in the course of the variola. The heart was large, weighing eighteen ounces and three-quarters; the right ventricle was hypertrophied and dilated; it formed the greater portion of the apex of the heart. Both the right chambers were quite full of decolorized fibrin, which adhered firmly to the internal surface of the auricle, thence passing through the tricuspid orifice into the ventricle. This was, no doubt, the immediate cause of death. The thrombus had been forming for some days previously, most probably from the 9th, when the unfavourable change above noticed took place, and afforded the only rational explanation of the subsequent symptoms and the sudden death.

The left auricle was dilated and thickened, and the orifices of the pulmonary veins were much enlarged. The left auriculo-ventricular opening was reduced to the size of the point of the index finger; it was funnel-shaped, opening into the ventricle by a slit-like aperture, and formed by the thickened segments of the mitral valve. The anterior segment was of cartilaginous consistence at the base, and the passage was smooth throughout; the papillary muscles and tendinous chords were not hypertrophied.

The aortic and pulmonic valves were healthy, and the left ventricle was strictly normal as to capacity and thickness.

The systolic bellows-murmur heard at the ensiform cartilage on the 4th of September, 1867, and repeatedly afterwards, but coinciding on every occasion with systolic pulsation of the jugular veins, was most probably due to tricuspid reflux. This is a phenomenon of very rare occurrence from simple engorgement of the right ventricle. The murmur of the same rhythm and quality which was subsequently developed at the apex, must

have resulted from mitral regurgitation. The displacement of the apex of the heart to the left was due to hypertrophy with dilatation of the right ventricle.\*

CASE CVII.—*History of Acute Rheumatism; Cough and Hæmoptysis; Orthopnœa and Œdema; Paroxysmal Dyspnœa; Irregularity of Pulse; Albuminuria; Epigastric Tenderness, and Extension of Hepatic Dulness; Pulmonary Œdema; Displacement of Apex of Heart Outwards; Presystolic Fremitus; Presystolic and Systolic Murmur at Apex; Presystolic Murmur likewise Audible at Left Margin of Sternum; Systolic Murmur at Xiphoid Cartilage; Death by Slow Asphyxia. Autopsy: Serous Effusion into the Pericardium; Enlargement of Heart; Thrombosis of Right Chambers; Dilatation and Hypertrophy of Right Auricle; Slight Hypertrophy of Right Ventricle; Thickening of Tricuspid Valve, and Constriction of Orifice; Dilatation and Hypertrophy of Left Auricle; Great Constriction of Mitral Orifice; Thickening of Papillary Muscles and Tendinous Chords; Left Ventricle Thinned at Apex, but otherwise Normal; Aorta Reduced in Size, Hyperæmic Internally, its Valves Thick and Slightly Incompetent.*

Patrick McD., aged twenty-three years, a shoemaker, was admitted into the Mater Misericordiæ Hospital on the 22nd of July, 1870. He had had rheumatic fever twice; viz., at the ages of nine and thirteen years respectively, and had been partially invalided for the last nine months. Five weeks anterior to the date of admittance he began to cough; he then spat blood for a week; three weeks later his feet began to swell, and for the last week dyspnœa had been so urgent that he could not lie down.

When admitted he was pale, but with a malar blush. He suffered from aggravated dyspnœa, which was occasionally paroxysmal. Respiration was 48. Pulse arhythmically irregular, varying from 96 to 108, and very weak; feet and legs greatly swollen; tongue clean and moist, but livid. Urine passed in small quan-

\* See *Proceedings of Pathological Society*, new series, vol. v., part i., p. 11.



tity, 1.020 in sp. gr., and containing a good deal of albumen. The liver was enlarged in both lobes, descending about two inches below the umbilicus, and tender to pressure. There was cedema of both lungs,

The apex of the heart pulsed in the sixth intercostal space, two and a-half inches outside the nipple-line. Here, a presystolic fremitus was perceptible; a coarse and jarring presystolic murmur was audible in the same situation, whilst the first sound was faint, and associated with a soft but feeble murmur.

At the xiphoid cartilage, a soft systolic murmur, somewhat metallic in quality, was heard; it was faintly propagated in the course of the aorta, but was not audible in the neck. The second sound was sharp at the base; it was not double, nor was it accentuated in the pulmonary artery. Tincture of nux vomica and quinine were prescribed, and the chest was dry-cupped. No improvement, however, took place, and the patient died on the 27th.

On examination of the body, half a pint of serum was found in the pericardium, together with a few flakes of unattached fibrin. The heart was enlarged, globular in figure, and weighed seventeen and a-half ounces; the visceral pericardium was thickened, opaque, and rough on the posterior surface of the right auricle, and to a slight extent also on the anterior surface of the right ventricle. Two long and pendulous flakes of fibrin adhered to the left apex. The root of the pulmonary artery was highly vascular and opaque on the external surface.

The right chambers contained a good deal of decolorized fibrin. It extended from the auricular appendix, which was filled with it, through the tricuspid orifice, and into the right ventricle; it did not, however, enter the pulmonary artery.

The tricuspid orifice was reduced to the size of the tip of the middle finger; it was ovoid in figure, and bounded by the coherent valve-segments. These latter were greatly thickened, but smooth and white, as were likewise the tendinous chords; the papillary muscles were thickened.

The right auricle was dilated and somewhat hypertrophied; the right ventricle was normal as to size, but thickened; the pulmonary artery and its valves were normal. The left auricle

was greatly dilated, and the segments of the mitral valve were united and thickened, and the mitral opening was converted into a slit which admitted only the point of the index finger; it presented some calcareous patches on the auricular aspect. The left papillary muscles and tendinous chords were likewise hypertrophied, but smooth. The left ventricle was altered only in a very slight degree; it was dilated and thinned at the apex.

The aorta was remarkably small in calibre; it was crimson-red internally; the aortic valves were slightly thickened and incompetent, but even and smooth on the surface.

CASE CVIII.—*History of Rheumatism; Sudden Right Hemiplegia and Aphasia; Rapid, Weak, and Feeble Pulse; No Cardiac Murmur; Death by Exhaustion. Extensive Softening of the Left Anterior Lobe of the Cerebrum; No Embolus Detected; Mitral Orifice Greatly Contracted by Cohesion of the Valve-Segments; Left Auricle Dilated and Thickened; Left Ventricle Normal.*

Jane Q., aged forty-seven years, was admitted into hospital on the 17th of March, 1866. She had had a few attacks of rheumatism, but otherwise she had been healthy up to the 28th December, 1865.

On the night of the 27th December she retired to rest in her usual state of health, and on the following morning it was found that she had lost the use of the right arm and leg, and of the corresponding side of the face; she had also lost the power of speech.

At the date of her admittance, there was complete motor paralysis of the right arm, and partial paralysis of the right leg; she dragged the leg in walking, and she was barely able to draw it up and extend it when in bed. The sphincters were not paralysed, nor was sensibility in either limb affected. The features were drawn towards the left side, and the tongue was protruded to the right.

The patient was, however, capable of closing both eyes, of corrugating the forehead, and of moving the tongue to the left side when directed to do so. She could swallow, and was

quite intelligent, but unable to utter a word beyond "yes" and "no." She moaned incessantly. The pupils were normal and equal in size. The pulse at the wrist could not be registered. The heart pulsated at the rate of 160, very feebly and irregularly. Both cardiac sounds were morbidly clear, and extensively transmitted over the chest; there was no cardiac murmur.

On the evening of the 20th of March, the right side of the face became flushed, its temperature rose, and it throbbed perceptibly, whilst the left side remained pale and cold.

She died on the 24th, having suffered much during the preceding few days.

The body was examined twenty-four hours after death. The brain was of normal consistence and apparently healthy, with the following exception: The anterior lobe of the left hemisphere was much reduced in volume, of a light-yellow colour, and semi-diffuent; its convolutions and sulci were effaced, and the left olfactory convolution and bulb were softened and enlarged, the former having become cylindrical, and been brought to the surface of the brain by obliteration of the sulcus. The brain exhibited no trace of hæmorrhage or of inflammation, and no plug was found in any of the cerebral vessels. The second and third left frontal convolutions were disorganized, as were likewise the left orbital convolutions.

The left auricle of the heart was enlarged and thickened; the mitral orifice was greatly contracted by cohesion of the valve-segments; it admitted only the tip of the index finger. The left ventricle was normal.

No cardiac murmur whatever was detected in this case. The patient was seven days under observation, but during the whole of that period her weakness was extreme.

CASE CIX.—*Palpitation and Cardialgia; Œdema; Hæmoptysis; Feeble and Irregular Pulse; Presystolic and Systolic Murmur at Apex of Heart, the former Suspended for Eighteen Days; General Serous Effusion; Death. Autopsy: Enlargement of Heart, and Fatty Growth on its Surface; Dilatation of Right Auriculo-Ventricular Orifice; Thrombosis of Right Chambers; Great Contraction of Mitral Opening; Dilatation and Hypertrophy of Left Auricle; Left Ventricle Normal; Aortic Valves Healthy and Competent; Extensive Sanguineous Infarction of the Lungs; Disfigurement of the Liver by Early Tight Lacing.*

Jane McG., aged fifty years, was received into hospital, June 1st, 1866. She had not had rheumatism. Two years previously, after suffering much anxiety, she experienced, for the first time, palpitation and pain in the region of the heart; her feet and legs had recently become swollen.

At the date of admittance, her condition was the following: Radial pulse not perceptible; slight œdema at outer ankles; extremities cold and livid; liver much enlarged and prominent, but even on the surface; it descended nearly to the umbilicus; she had not had jaundice; respiratory sounds were normal, with the addition of coarse muco-crepitus over base of right lung. The action of the heart was remarkably irregular, and at the rate of 150 in the minute; impulse strong and labouring, thus contrasting very strikingly with the radial pulse. Both cardiac sounds were clear and ringing; they were extensively transmitted over the front of the chest, especially in the course of the aorta, and were unattended with murmur.

On the following day the pulse was perceptible at the wrist, but less distinct on the right than on the left side; it was 84 in the minute. Ordered a tonic, consisting of iron and chloric ether, with infusion of quassia.

June 10th. Œdema of lower limbs much increased; orthopnoea; congestion of base of left lung. To have, every sixth hour, a draught consisting of tincture of digitalis, ℞; spirit of



juniper, 3j; and camphor water, 3j. To be dry-cupped over left base.

On the 15th, a wiffing systolic murmur was heard at the left apex; it was not audible elsewhere. The second sound was intensified in the pulmonary artery. The base of the left lung was dull, and yielded feeble respiratory sound with crepitus. There had been slight hæmoptysis in the course of the preceding night. The legs were somewhat swollen, and the patient complained of a stabbing pain below the left nipple. To have an opiate plaster applied there.

On the 19th, a grating murmur was heard at the apex; it preceded the first sound immediately, occupying the latter part of the diastolic pause. Four days previously I had inferred, from the general condition of the patient, the *probable* existence of mitral narrowing, and mentioned my impression to the clinical class; but I did not venture upon a positive diagnosis in the absence of the pathognomonic murmur, which was only heard for the first time on the 19th. On that day an affirmative diagnosis of mitral contraction was made.

On the 21st, the pulse-rate being 114, the respiration 30, and the action of the heart very irregular, a systolic murmur was alone heard at the apex. On the 22nd, this was no longer audible, whilst a harsh presystolic murmur was heard at intervals. The heart acted irregularly, both in regard to force and rhythm; it contracted spasmodically and with much energy several times in rapid succession, every three or four minutes; in the intervals it acted feebly. Coincidentally with the strong contractions, the presystolic murmur was heard; but it was inaudible during the period of feeble action.

On the 23rd it was very distinct, and on the 25th it was again all but suppressed.

27th. Copious expectoration of dark unmixed blood; presystolic murmur faintly audible. To have ℥x of dilute sulphuric acid in 3j of water every third hour.

28th. Great respiratory distress, and evidence of effusion on both sides of the chest; pulse 162; hæmoptysis continued. She died on the following day.

The body was examined by my resident clinical clerk, Mr.

O'Hanlon, eight hours after death. Both pleuræ and the pericardium contained a large quantity of serum. The left lung presented, on its anterior edge, a nodule of sanguineous infarction, as large as a walnut, and on its outer surface a small mass of cretified tubercle. There were, in the inferior lobe of the right lung, several large collections of solid and extravasated blood, and in the superior lobe numerous but smaller infarctions, so that the surface of section presented a mottled appearance.

The heart weighed twelve ounces and three-quarters. There was a good deal of fat upon its surface, especially around the roots of the great vessels. The right auricle contained some dark blood-clot; the right ventricle was slightly dilated but much thickened, and the tricuspid orifice so much dilated as to permit the passage of four fingers placed in juxtaposition. Entangled in the columns of the anterior wall of the ventricle was a large flake of solid yellow fibrin, whence it extended into the pulmonary artery for a distance of two inches.

The left auricle was much dilated and hypertrophied; the appendix was thickened in a high degree, and the orifices of the pulmonary veins were greatly dilated; the superior and left admitting the index and middle fingers. The mitral orifice barely admitted the point of the index finger passed from the auricle. It presented the appearance of a narrow slit, with thick, smooth, and well rounded edges. Both segments of the valve had become rigid; the anterior and right, thicker than the posterior and left segment, presented a smooth but uneven surface towards the ventricle, quite to its root at the aortic opening; both it and the attached tendinous chords were white and glistening, and one of the latter, which was connected with the anterior fleshy column, was as thick as the little finger. Both segments, though thick and firm, were flexible, and fell together so as to close the opening, but not perfectly. When the ventricle was filled with water, the aorta having been compressed with the fingers, there was a slight leakage into the auricle, owing to the maladaptation of the edges of the valves. The left ventricle was normal. The aortic valves were healthy and competent. The aorta was dilated, and in the ascending and transverse portions of the arch it was somewhat patchy on the internal surface. The liver exhibited the effects

of tight lacing in early life ; it was contracted in its transverse diameter, and elongated vertically ; on its anterior surface there was a deep horizontal sulcus, corresponding to the edge of the united costal cartilages, the adjacent portion of the capsule being thick and opaque.\* The suppression of the characteristic murmur of mitral narrowing for a period of eighteen days, and its subsequent unsteadiness, demand special notice. The extreme weakness of the patient during this entire period, affords, in my judgment, a rational explanation of the circumstance. This view receives support from the observation made on the 22nd of June ; namely, that the murmur was heard during the period of vigorous, though spasmodic action of the heart, but was inaudible in the intervals, when the heart acted feebly.

CASE CX.—*Rheumatism, and Hepatic Disease ; Dyspnœa, Hæmoptysis, and Œdema ; Congestion of both Lungs, and Cough ; Pulse Small but Regular ; Increase of Precordial Dulness ; Presystolic and Systolic Murmur at the Apex ; Second Sound Double ; Pregnancy at the Eighth Month ; Premature Confinement ; Death. Autopsy : Enlargement of the Heart by Hypertrophy and Dilatation of Left Ventricle ; Thrombosis of Right Chambers ; Partial Stenosis of Tricuspid Opening, and Thickening of Tricuspid Valves ; Great Dilatation, and Partial Hypertrophy of Left Auricle ; Stenosis of Mitral Orifice by Cohesion of Valves ; Calcification of Auricular Surface of Mitral Valves and Adjacent Endocardium ; Dilatation and Hypertrophy of Left Ventricle ; Stenosis of Aorta.*

Sarah M., aged twenty-six years, married, and in the eighth month of pregnancy, was admitted July 20th, 1869. She was the wife of a soldier, and had spent three years in India. Whilst there she had rheumatic pains and "liver complaint," the latter, however, unaccompanied with jaundice.

Eight months prior to admittance, she began to suffer from shortness of breath ; five months later, she repeatedly spat blood, and three weeks afterwards her feet became swollen.

State on admission : Feet, legs, and thighs greatly swollen,

\* See *Medical Press and Circular*, July 3rd, 1866.

but pale. Pulse 96, small, regular, and equal at both wrists; loud and dry cough; both lungs congested posteriorly, but the right in a greater degree than the left. Respiration 36, greatly embarrassed, and gasping; face pale, but with a malar blush, and congestion of lips. Urine scanty, 1·017 in sp. gr., and free from albumen. Precordial dulness was somewhat increased in extent. At the point of apex-pulsation, which was in the nipple-line and fifth intercostal space, a loud presystolic murmur was heard, and immediately succeeding it, a soft systolic murmur. At the base, a systolic murmur was heard faintly, and by transmission. The second sound was double, and heard all over the precordium.

The pulsation of the foetal heart was detected near the umbilicus; it was slow, exceeding the rate of the maternal heart in a slight degree only.

The chest was dry-cupped and poulticed, an aperient was given, and subsequently an active and stimulant diuretic.

She was prematurely confined of a still-born child on the 21st, after a labour of ten hours' duration. Slight uterine tenderness, which was accompanied by a fetid discharge from the vagina, was detected on the 24th. The breathing was much relieved by the evacuation of the uterus, but the cardiac signs were as previously noted.

On the following day the extremities were cold, and she spat some blood; in the evening she began to sink, and at 5 P.M. on the 26th, she died.

The body was carefully examined, with the following result: The heart was very large, and weighed nineteen and a-half ounces; there was a good deal of fat upon its surface, and, owing to hypertrophy of the left ventricle, the apex was magnified and blunted.

The lining membrane of the aorta, pulmonary artery, and pulmonary veins, was hyperæmic, and of a deep crimson tint. The right chambers contained a large decolorized thrombus, which was engrained into the pectinate wall of the auricle, passed thence into the ventricle, where it was entangled by a number of filaments with the tricuspid valve and the columnæ carneæ, and from the ventricle it extended by a large cylinder into the pulmonary



artery. The thrombus was hollow, constituting a thick and smooth lining to the chambers, and through it the circulation must have been carried on during the last hours of life. The right auriculo-ventricular orifice was contracted, admitting only the tips of two fingers; the segments of the tricuspid valve were thick, but apparently not incompetent. The right auricle and ventricle were normal as to capacity and thickness of walls. The left auricle was greatly dilated, and in a slight degree thickened; it was full of dark clotted blood. The lining membrane of the auricle, to the extent of half an inch around the mitral orifice, was rough and spiculated by deposition of a nearly uniform layer of calcareous matter.

The segments of the mitral valve were greatly thickened, and agglutinated together so as to form a circular curtain, rough and cretified on the auricular surface, but uniformly smooth and free from lime-salts on the ventricular side. In the centre of this curtain, which was rigid and immovable, there was an opening which was transversely oval, smooth on the edges, and barely large enough to admit the point of the index finger. The connected tendinous chords were much thickened, and the papillary muscles and the fleshy columns generally were hypertrophied. The left ventricle was dilated and greatly hypertrophied. The valves of the aorta were thick, but pliant, smooth, and competent, and the orifice was reduced to the diameter of the point of the little finger.

The quantity of blood which, in this case, passed into the aorta at each systole of the left ventricle, must have been very small, owing to the twofold lesion of mitral obstruction and incompetency. Hence, the extreme contraction of the aortic orifice, and notwithstanding this condition, the absence of a murmur of aortic obstruction. The opposite characters of the two murmurs heard at the apex of the heart, the harsh and grating quality of the presystolic, and the soft and blowing character of the systolic murmur, were alike explained by the conditions of the corresponding surfaces of the mitral valve. Hypertrophy and dilatation of the left ventricle were due, primarily and mainly, to inadequacy of the mitral valve.

CASE CXI.—*No History of Rheumatism ; no Hæmoptysis ; Recurrent Bronchitis ; Pallor and Debility ; Rapid but Regular Pulse ; Tumultuous Action of Heart, and Projection of Precordium ; Presystolic Fremitus and Murmur ; Postsystolic Musical Note ; Improvement ; Sudden Right Hemiplegia and Aphasia ; Death. Autopsy : Plugging of Left Anterior and Middle Cerebral Arteries ; Enlargement of Heart ; Thrombosis of Right Chambers and Pulmonary Artery ; Dilatation of Right Ventricle ; Dilatation and Hypertrophy of Left Auricle and Ventricle ; Extreme Stenosis of Mitral Orifice by Cohesion and Thickening of Mitral Valves ; Attachment of a Movable Thrombus to Anterior Segment of Mitral Valve ; Narrowing of Aorta, and Thickening and Inadequacy of Aortic Valves.*

Robert F., aged seven years, an inmate of a reformatory, was admitted into the Mater Misericordiæ Hospital on the 5th of November, 1872. He had not had rheumatism, nor had he spat blood. When admitted he was suffering from bronchitis. On examining him the following day, I found his condition to be as follows: He was pale and emaciated, and evidently had been indifferently nourished; he coughed, was feverish, and the pulse was rapid (120), but regular. The precordium projected considerably, especially on the left side, and the heart pulsated tumultuously. The apex-pulsation was felt in the fifth intercostal space, half an inch inside the nipple-line. At this point a presystolic fremitus was readily detected, and from this sign, without proceeding further with the examination, the diagnosis of mitral narrowing was at once made. A presystolic murmur likewise existed at the apex.

Both sounds of the heart were remarkably clear and sharp; but succeeding the first sound, and appended to it, was a musical note, which was likewise distinctly audible in the left axilla and back.

He improved under ordinary treatment, the bronchitis was less troublesome, and in the course of four or five days he was allowed to leave his bed and walk about the ward. By some

misadventure he contracted fresh cold, and had another attack of bronchitis which was more severe than the former.

He was confined to bed on the 18th and 19th, and exhibited greater debility and respiratory distress.

On the evening of the 19th his condition had undergone some improvement, and he declared that he felt better. He was seen by the Sister in attendance at 10 and 11 o'clock, respectively, when he seemed better. On the following morning he was found to be quite paralyzed on the right side, and unable to utter a word.

When I saw him at 11 o'clock on the 20th, he was completely paralyzed as to motion in the right arm and leg; but sensibility was in these limbs rather exalted. The features were slightly drawn to the left; the pupils were normal and equal, and there was no strabismus. He perfectly understood what was said to him, and in the effort to reply, his features were drawn more to the left side; he could not utter a word save "yes" and "no," and even these were expressed very indistinctly.

The pulse was rapid, the skin was hot and dry, and loud bubbling crepitation was heard all over the chest. He either could not or would not protrude his tongue; but deglutition, as regarded liquids, was perfect. The tactile and acoustic signs at the precordium were those previously noted.

He died on the evening of the 20th, evidently from accumulation of mucus in the air-passages, and the body was carefully examined by Dr. Nixon on the following day.

The brain was large, and nowhere softened; the arteries at the base were empty, except the left anterior and middle cerebral, both of which were plugged to distension with small and unequal sized particles of solid fibrin. The terminal portion of the carotid, at the proximal side of the obstruction, was empty; but the branches arising from both the infarcted vessels, at the distal side, were full of dark liquid blood.

The bronchial tubes throughout were congested, and contained much blood-stained frothy mucus. The heart was somewhat enlarged; it was globular in figure; the right chambers contained some dark soft coagulum, and a few detached masses of fibrin;

the former extended into the pulmonary artery, and was there coated with a film of fibrin.

The right ventricle was dilated and thinned; the left auricle and ventricle were dilated and hypertrophied, and the cavity of the left ventricle was rounded.

The aorta, including the orifice, was reduced in calibre, and the aortic valves were thick and puckered, but pliant; they were inadequate to close the orifice, as shown by the water-test.

The mitral orifice was constricted in a high degree; it admitted the point of the index finger with some difficulty; its figure was that of a button-hole, and it was bounded by the valve-segments, which were smooth and thick on the edges, being elsewhere fused into an elliptical curtain. To the anterior and right portion of this curtain a mass of solid fibrin was attached. This body was of the size and shape of a musket ball, but rough and shreddy on the surface; it involved, in its substance, about one-half the depth of the valve, and projected into the opening in such a way that it must have been wafted across the orifice of the aorta by the stream of blood entering the ventricle from the auricle. The emboli found in the cerebral vessels were, no doubt, derived from the jagged surface of this mass of fibrin by attrition of the blood-stream.

I cannot, with equal confidence, attribute to the vibration of this body in the mitral orifice at the acme of ventricular systole, the musical note of postsystolic rhythm which was heard at the apex and in the left axilla and back on the 5th of November, and repeatedly afterwards. My difficulty arises from the consideration that such a mode of origin would necessarily imply, either a murmur of mitral regurgitation, which did not exist, or, assuming that the body prevented reflux by acting as a ball-valve, the impossibility of its freely vibrating whilst so acting.

The case illustrates, firstly, the not unfrequent absence of a record of rheumatism in the history of mitral stenosis, even in the child; secondly, the precipitation of fibrin from the blood whilst still in circulation, owing to the twofold cause of partial stasis, and obstruction by a rough or uneven obstacle; and lastly, the detachment by the circulating blood, of detritus from the surface



of the thrombus so formed, its impaction in the left middle and anterior cerebral arteries, by which the left motor centre and Broca's region of speech were deprived of arterial blood, and right hemiplegia and aphasia were produced.\*

CASE CXII.—*Acute Rheumatism ; Strain of Right Arm, and Aneurism by Rupture of the Ulnar Artery ; Palpitation ; Sudden and Complete Right Hemiplegia, with Partial Aphasia ; Visible and Collapsing Pulse ; Pulsating Tumor on Front of Right Forearm ; Dyspnoea and Hæmoptysis ; Œdema ; Double Basic, and Apex-Presystolic Murmur ; Death. Autopsy : Clot in Left Hemisphere of Brain ; Great Enlargement of Heart ; Disorganization of Aortic Valves ; Contraction of Mitral Orifice, with Deposit of Beaded Lymph upon Auricular Surface of Mitral Valve ; Fatty Degeneration of Heart, and Fatty Condition of Liver ; Large Aneurism of Right Ulnar Artery.*

Michael C., aged twenty-three years, a wire-worker, was admitted under Dr. Nixon's care, in September, 1872. By Dr. Nixon's kind permission I examined the man and took notes of his case.

The patient's habits had been temperate, but he had had three attacks of acute rheumatism. Twelve months previously, after working for an entire day with a pair of blunt pliers, which required powerful grasping pressure with the hand, he felt numbness and tingling in the three inner fingers of the right hand, which was the one he had been using. Shortly afterwards he noticed, on the inner portion of the anterior aspect of the right forearm, a swelling, which "panted"; this swelling continued to be of the original dimensions up to the date of his admittance.

He had been, for some time, subject to palpitation on exerting himself; and six weeks prior to admission he suddenly experienced giddiness, and, at the same time, lost the use of his right

\* See *Proceedings of Pathological Society*, vol. v., part ii., new series, p. 118.

arm and leg ; his speech was likewise, but only partially, affected, but feeling remained perfect in the paralyzed limbs.

When I saw the man, his right arm was quite powerless ; his right leg was likewise paralyzed, but in a less degree ; whilst in both, sensibility was unimpaired. He was pale ; the features were slightly drawn to the left ; articulation was thick and slow, but the tongue was protruded in the axial line. The pulse was about 84, visible and collapsing, but it was regular and equal on both sides. A swelling existed on the ulnar portion of the front of the right forearm, an inch and a-half below the internal condyle of the humerus ; it yielded a pulsation, which was full and expansile, and synchronous with the radial pulse. Pressure on the brachial artery above the condyle stopped the pulsation in the tumor, and direct pressure entirely removed the swelling. If, whilst the hand remained on the tumor, pressure were suddenly withdrawn from the artery above, pulsation was at once restored in the former, and a feeling of sudden influx, accompanied by thrill, was communicated to the hand.

At midsternum, and likewise at the apex, but by transmission, two murmurs were heard. Of these, one was faint, and systolic in time, whilst the other was loud and whiffing, and *succeeded* a nearly normal second sound ; the former alone was audible in the arteries of the neck.

On again visiting the man on the 25th of October, I found that he had regained all but perfect use of the right limbs, and entirely recovered the power of articulation. The ulnar aneurism had undergone slight enlargement. The physical signs were as previously described, with this addition, that a faint presystolic murmur now existed at the left apex. He was shortly afterwards discharged.

The man was readmitted for Dr. Nixon, on the 25th of January, 1873, and I was again favoured with the opportunity of examining him. He was then greatly changed ; the face and neck were puffed and congested, respiration was greatly embarrassed, and the feet and legs were swollen.

The ulnar aneurism seemed to me unchanged, but the aortic murmurs were both much more harsh ; the apex presystolic murmur was more distinct. He had hæmoptysis accompanied

by great dyspnœa on the 3rd of February, and he died on the 4th.

Autopsy : The heart was greatly enlarged, and weighed twenty-six ounces ; the right ventricle was thin and dilated ; it exhibited dun mottling on its internal surface, and the portions so changed in colour were found to have undergone complete fatty transformation. The tricuspid and pulmonic orifices and valves were normal. The left auricle was dilated and somewhat thickened ; its lining membrane was opaque. The mitral opening was narrowed to the size of a florin piece, and the mitral valves were studded, on the auricular aspect and near the attached margin, with a series of fibrinous nodules, as large as millet seeds, and arranged linearly ; the valves were otherwise normal, and were competent. The left ventricle was greatly dilated and thickened ; it was likewise mottled of a dusky yellow internally, and where these spots existed, it was structurally identical with the discoloured portions of the right ventricle.

The aortic valves were utterly disorganized ; they were rolled up into coils, were rough and friable, and beaded with nodules of fibrin.

The aorta was slightly atheromatous. A large blood-clot, nearly deprived of colour, was found in the medullary substance of the left cerebral hemisphere ; it had descended into the lateral ventricle, where it rested on the outer side of the corpus striatum. A small branch of the left middle cerebral artery had been closed, and converted into a fibrinous cord.

The liver cells contained a good deal of oil, and many of the muscular fibres of the heart were in an advanced state of fatty change. An aneurism existed upon the right ulnar artery, one inch and a-half below the bifurcation of the brachial.

In reference to the condition of the mitral orifice, Dr. Nixon made the following judicious remarks when reporting the case and exhibiting the diseased parts before the Pathological Society of Dublin.\* “The beads of lymph found on the auricular surfaces of the mitral valves were certainly of themselves sufficient in amount to produce obstruction to the passage of the blood into

\* See *Proceedings*, vol. v., part ii., new series, page 137.

the ventricle; and I think from the dilated condition of the latter, the large reflux of blood from the aorta must have brought the mitral curtains towards each other, thus exposing their roughened auricular surfaces to the passage of the blood, and thus producing the obstructive murmur."

No doubt, the premature distention of the ventricle by the sudden entrance of a large volume of blood from the aorta was, by depressing the valves, a contributory, though only a secondary, cause of the presystolic murmur, and must have intensified it considerably in the last days of the man's life.

CASE CXIII.—*Repeated Attacks of Rheumatism; Intemperate Habits; Hæmoptysis; Œdema; Hepatic Engorgement and Tenderness; Quick and Weak Pulse; Outward Displacement of Apex; a Presystolic and a Systolic Murmur at Apex-Point; Harsh Systolic Murmur at Right Base, and Transmitted into the Carotid Arteries; a Superficial Systolic Grating at Left Base; Second Sound Loud and Sharp; Sudden Loss of Power in Right Hand and Forearm, and soon afterwards, Sudden Death. Autopsy: Congestion of Lungs and Liver; Adhesion of Pericardium, and Enlargement of Heart, with a Cheese-like Mass Imbedded in its Anterior Surface; Thrombosis of Right Chambers; Dilatation of Right Ventricle; Dilatation and Hypertrophy of Left Auricle and Ventricle; Extreme Narrowing of Mitral Orifice, and Calcareous Conversion of Mitral Valves; Calcareous Change, but without Incompetency, of Aortic Valves.*

Thomas W., aged thirty-six years, a blacksmith, of very intemperate habits, was received into hospital, May 30th, 1873. He had had several attacks of rheumatism, the first at the age of sixteen years. His breathing had been short for the last twelve years. One year prior to admission he began to spit blood, and hæmoptysis had recurred at intervals since that date. His feet had been swollen for the last month.

State on admittance: The back was arched, the chest narrow, and the sternum prominent. Pulse very weak but regular, and 108. Throbbing of the carotids, and cedema of the lower limbs.



He complained of pain in the left side of the chest from the clavicle downwards, and spat blood daily in large quantity. Breathing not much embarrassed; occasional cough; features sunken. The kidneys acted indifferently; the urine was loaded with lithates, but was otherwise normal; the liver was engorged, and tender to pressure, and the bowels were inflated; the impulse of the heart was feeble; the apex-point was in the nipple-line, and there a presystolic and a loud systolic murmur were heard in unbroken continuity. The initial portion, corresponding to the presystolic constituent of this prolonged and compound murmur, was readily distinguished from the latter or systolic portion, by its being harsh and emphasized, and by its obviously preceding the apex-pulsation.

At the right base, a rough systolic murmur existed; it was faintly transmitted into the arteries of the neck. At the left base, a coarse and superficial rub of systolic rhythm was heard. The second sound at the base was loud and sharp, but no diastolic murmur existed.

On the night of the 2nd of June, he suddenly lost the use of the right hand and forearm, which then became gradually swollen; yet no difference between the radial pulses could be perceived. There was total loss of rest. To have six leeches at the epigastrium, and ℞v of Battley's sedative at night. His condition underwent no marked improvement, and he died suddenly on the 12th.

Autopsy, twelve hours afterwards: The lungs were voluminous, and engorged with blood; the bases were solid, and dark in section. The anterior surface of the heart, and the corresponding portion of the parietal pericardium, were roughened by effused lymph, and the opposed surfaces were connected by a few long bands of fibrin. In the anterior surface of the right ventricle, and surrounded by adhesion, a cheese-like mass, about as large as a pea, was imbedded. The heart weighed twenty and a-quarter ounces; there was a good deal of fat on its surface, chiefly in the grooves. The aorta was of normal size, and free from disease. The right chambers contained a mass of solid fibrin, which extended into the pulmonary artery; the right ventricle was dilated and thinned; the valves on the right side of the heart were

healthy. The left auricle was greatly dilated and hypertrophied, and its lining membrane was thick and opaque; the left ventricle was dilated and much thickened. The mitral orifice was reduced to the diameter of the point of the little finger; it was of a button-hole shape, and its valves were shrunken and converted into a calcareous but friable mass, which was remarkably rough on the surface; the anterior and right segment was more disorganized than the posterior and left.

Both the anterior segments of the aortic valve were converted into rugged, calcareous masses, which projected into the orifice; but the posterior segment was soft and pliant, and so much expanded that it overlapped the diseased segments, covering the axial passage left between them so as to prevent regurgitation. The orifices of the coronary arteries, and the vessels themselves, were unaltered. The liver was large and congested; the kidneys were healthy.

CASE CXIV.—*Rheumatism; Intemperance; Hæmoptysis and Dyspnœa; Anasarca and Ascites; Albuminuria; Weak Pulse; Jugular Pulsation; Systolic Murmur, accompanied by a Musical Note, at the Apex of the Heart and over the Body of the Ventricle; Feet Punctured, and Rapid Dispersion of Dropsical Effusion; Gangrene of Feet and Legs; Asystole; Death. Autopsy: Serous Effusion into the Peritoneum; Enlargement of the Liver and Spleen; Kidneys Enlarged, and in Transition Stage to Atrophy; Lungs Emphysematous; Heart Enlarged, and Adherent to Pericardium; Right Chambers Hypertrophied and Dilated; Tricuspid Orifice Dilated, and Valves Thickened; Left Chambers Dilated and Hypertrophied; Mitral Orifice greatly Contracted, and Valves Calcified; Aorta Dilated and Atheromatous; Pulmonary Artery Dilated.*

Matthew N., aged fifty-eight years, a sailor, was received into hospital, August 25th, 1874. At the age of seventeen he had rheumatic fever which lasted three months; had been a hard drinker, and had repeatedly spat blood. Two years prior to date of admittance he had an attack of bronchitis, and then for the first time noticed his breathing to be very short. His feet had

become swollen within the previous six weeks, and when he was admitted the following was his condition. The lower limbs were greatly swollen; there was some liquid in the peritoneum; respiration was embarrassed; pulse 108, and very weak, but regular; urine scanty, 1.025 in sp. gr., acid, and containing some albumen and blood. The external jugular veins were full, but not distended; they pulsated synchronously with ventricular systole. There was comparative dulness with crepitation over the base of the right lung. The area of precordial dulness was contracted, owing to an emphysematous condition of the edges of the lungs. The apex-pulsation could not be distinctly felt; but, two inches below the nipple and in the fifth intercostal space, a systolic bellows-murmur of high pitch, and terminated by a sharp musical note, was audible. This murmur was still more distinctly heard in the fourth intercostal space, midway between the nipple and the sternum, and it was faintly audible for a distance of two inches upwards and to the right; it could not be heard in the axilla or in the back. In the course of a few days it became necessary to puncture the feet in order to avert gangrene. A copious flow of serum from the punctures took place. Diarrhœa, accompanied by diuresis, set in, and under the combined influence of this twofold drain, the œdema of the lower limbs and the ascites were entirely dispersed in less than forty-eight hours. The pulse was now 96, very weak, slightly irregular and faltering. The following tracing (Fig. LX.), taken by Dr. Nixon, exhibits these characters.

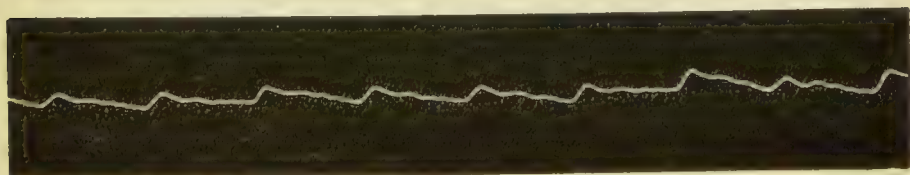


FIG. LX.

Mitral constriction and tricuspid regurgitation.

Matthew N. Pulse 96. September, 1874.

For a week subsequently the diarrhœa continued at the rate of about twenty motions in the twenty-four hours, and the discharge from the punctures was kept up. During this period, de-



spite the frequent calls to the night-chair, the poor man experienced the greatest relief; he could sleep in the recumbent posture, and was quite free from suffering. He had been taking, thrice daily, ℥v of tincture of digitalis with ℥x of tincture of the perchloride of iron; this was now suspended.

On the 15th of September, diarrhœa having for some days ceased, anasarca and ascites began to re-appear. A repetition of puncturing soon afterwards became necessary; but, despite this measure, gangrene of the feet and legs followed, and after three days of great suffering, during which no pulse could be felt at the wrist, the patient died on the 2nd of October. The body was examined on the following day.

*Abdomen:* The peritoneum contained several quarts of serum; the liver was enlarged, and in an early stage of cirrhosis; it weighed four pounds seven ounces, was rough on the surface, firm in texture, and its interstitial fibrous tissue was hypertrophied; the gall-bladder was distended with bile. The spleen weighed eleven ounces, was firm, and its capsule was thickened. The right kidney weighed seven ounces, and the left kidney six ounces; they were both firm in texture, light-coloured in section, and the tubes were loaded with detached epithelium; they were manifestly in the transition-stage, from hypertrophy to atrophy, of the "large white kidney."

Peyer's patches were somewhat magnified, but there was no ulceration or hyperæmia of the bowels.

*Thorax:* The lungs were voluminous and emphysematous. The heart was enlarged, globular in figure, and firmly attached to the pericardium by old adhesions on the anterior and posterior surface; it weighed nineteen ounces. Both right chambers were hypertrophied and dilated; they contained a good deal of blood-clot. The tricuspid orifice was greatly magnified, and the tricuspid valves were somewhat thick, opaque, and fringed with red; they were incompetent owing to dilatation of the orifice. The left auricle and ventricle were dilated and hypertrophied, and the orifices of the pulmonary veins were much enlarged. The mitral opening was greatly contracted; viewed from the ventricle, it was of the size and figure of a coat button-hole, somewhat rounded at the anterior extremity, and larger



than at the posterior. On the auricular aspect it presented a crescentic figure, convex anteriorly. The valve-segments were thick and perfectly rigid; they were universally calcified, but, being invested by the endocardium, they were smooth at all points except near the edge and on the auricular surface, where they were slightly spiculated. The attached tendinous chords were attenuated, and quite free from calcific change. The aorta was dilated above the valves and throughout the arch; it was in an early stage of atheroma. The aortic valves were healthy and competent, as were likewise those of the pulmonary artery. The last mentioned vessel was dilated, but its valves were competent. The muscular structure of the heart was healthy.

The chest had been repeatedly examined, and the diagnosis arrived at was, Mitral and tricuspid regurgitation, most probably from cartilaginous thickening of the valves, with fibroid transformation of the papillary muscles and inner layer of the ventricular walls. Presystolic murmur was not at any time detected; and, as the heart was carefully and almost daily examined during the thirty-nine days of the man's residence in hospital, I am satisfied such could not have existed. During the entire of this period the pulse was weak and small, but not faltering, or irregular.

The case, then, illustrates protracted suspension of presystolic murmur. This, I believe to have been due to debility of the left auricle, arising from general weakness.

CASE. CXV.—*History of Cardiac Disease of Long Standing; Repeated Hæmoptysis; Orthopnœa; General Dropsy and Cyanosis; Increased Area of Precordial Dulness; Presystolic Murmur at the Apex, and also at the Left Margin of the Sternum; Gangrene of the Feet and Legs; Diagnosis of Mitral and Tricuspid Narrowing; Death. Contraction of both Auriculo-Ventricular Orifices.*

Margaret M., aged twenty-five years, unmarried, was sent to me by Dr. Finegan, medical officer to the Finglas Dispensary, and admitted into the Mater Misericordiæ Hospital on the 11th of March, 1874. She had had measles of a bad type at the age

of sixteen. During convalescence, she caught cold from premature exposure, and was never subsequently in good health; her breathing became short, and she was subject to dizziness and palpitation.

Five years prior to admittance she spat some blood, and she had repeatedly spat blood subsequently. Menstruation ceased in December of the preceding year. Three weeks before admission and coming under my notice, her feet began to swell, and there was, at the same time, a decrease in the renal secretion. The following was her actual condition on the 11th of March. There was venous congestion of the face, and great turgescence of the veins of the neck; general lividity and depressed temperature of the surface; great œdema of the lower extremities; oppression of breathing, and orthopnoea. There was constant teasing cough, with slight frothy expectoration. The pulse was rapid, and so feeble that it could not be counted at the wrist. There was evidence of œdema of the bases of both lungs, and of effusion into the peritoneum. Precordial dulness was extended towards the right side; the impulse of the heart was strong and rather heaving, and the apex pulsated to the left of the nipple-line. Over the apex of the heart a well pronounced presystolic murmur of the ordinary character was heard; it was rough, but not remarkably so, and it extended quite up to the first sound.

In addition, there was audible to the left of the sternum, over the inner portion of the fifth costal cartilage and the corresponding part of the fourth intercostal space, a murmur of the same rhythm, but much more harsh than the former, and extending farther backwards into the long pause. At a point intermediate to the seats of these two murmurs, neither was distinctly audible.

The last mentioned circumstance I considered of so much significance, that upon it mainly, I ventured to make the diagnosis of obstruction at both auriculo-ventricular orifices. I repeatedly stated this opinion in presence of the clinical class, and I also mentioned it to my colleague Dr. Nixon whilst discussing the case with him. The poor woman's condition admitted of alleviation only. Gangrene of the feet and legs occurred, and she

died exhausted on the 14th of March, four days after her admission.

On examination of the body, the lungs were found to be congested and cedematous. There was some liquid in the pericardium. The heart was somewhat globular in figure, and weighed eleven ounces. The right auriculo-ventricular orifice was so reduced in diameter as to admit only the point of the middle finger; the tricuspid valves were slightly thickened, and united at the angles. The right auricle was much dilated and hypertrophied. The right ventricle was not dilated; it contained a good deal of blood-clot and decolorized fibrin, part of which was entangled in the tricuspid opening. The mitral orifice was still more contracted than the tricuspid, admitting only the point of the index finger.

The left auricle was thickened and dilated in a high degree, and the orifices of the pulmonary veins were dilated.

The left ventricle was not dilated or hypertrophied; it was remarkably thin at the apex, near the septum.

The point of greatest diagnostic importance in this case was the existence of two centres of presystolic murmur; one occupying the usual situation at the apex of the heart, and the other corresponding to the sternal end of the fifth costal cartilage and fourth intercostal space on the left side. Around these points, and within a circle of about two inches and a-half in diameter, the murmurs were respectively audible; but between and at an equal distance from them, there was a space where neither murmur could be distinctly heard.

This constituted the principal element on which the differential diagnosis was based. Corroborative evidence was, however, afforded by the difference in quality and in length of the murmur heard at the two points mentioned, a difference which I incline to attribute to the entanglement of fibrin in the edges of the tricuspid valves; and also by the existence of general venous turgescence and cyanosis. This latter condition is so exceptional in cases of mitral constriction, that, if copious effusion into both pleuræ were not likewise present, I should regard it as presumptive evidence of the existence of the double lesion.

In none of the published cases of mitral and tricuspid narrow-

ing combined, was the second sound reported to have been accentuated or doubled; a circumstance to be attributed, in my judgment, to the fact that, as regarded obstruction in front, and defective supply from behind, the two ventricles were in a similar predicament. The contracted auriculo-ventricular orifices served, each, as a point of resistance to the opposite ventricle, and as a stop to the corresponding ventricle. Hence, the absence of unequal dilatation and hypertrophy of the ventricles, and of doubling and intensification of the second sound.\*

CASE CXVI.—*Œdema of Feet; Frequent and Copious Hæmoptysis; Rheumatic Pains; Congestion of Face, and Exophthalmia; Dyspnœa and Cough; Headache and Recurrent Syncope; Pain in Region of Heart extending down the Left Arm, and in both Scapular Regions; Congestion of Right Lung and of Liver; Heaving Impulse at Precordium; Apex-pulsation much to the Left of Nipple Line; Postdiastolic Murmur at Apex; Subsequent Development of Presystolic Murmur, which was twice afterwards Suppressed and Restored alternately; Fremitus; Accentuation of Second Sound; Patient Six Years under Observation. Diagnosis: Progressive, and now Extreme Mitral Stenosis; Great Dilatation of Right Ventricle, and Consequent Displacement of the Apex to the Left Side; Exophthalmic Goitre.*

Mary E. C., aged twenty-five years, a machine sewer, consulted me for the first time in the summer of 1868. Of this visit I have preserved no record beyond the date. Her second visit was made on the 30th of June, 1869. On the latter occasion, I learned that for the last two months the feet had exhibited slight cedema in the evening, and that she had spat some blood. Has teasing dry cough; breathing much oppressed; pulse 120, small, and regular; face florid, eyes prominent, and thyroid body somewhat enlarged, but no distention of the cervical veins. She has frequently suffered from headache, and has constant sharp pain in the left side below the nipple; cannot lie on that side,

\* Case 61 (p. 710) completes the list of fifteen fatal cases of mitral stenosis in which the body was examined after death.



or sleep in the recumbent posture. Cardiac impulse heaving, and apex-pulsation half an inch outside the left nipple-line. The sounds of the heart were normal at the base, but at the apex a loud bellows-murmur of postdiastolic rhythm was heard. The second sound was accentuated in the pulmonary artery. To have ether and ammonia in camphor water.

October 5th. Pain very severe in the region of the heart, and extending down the left arm, as far as the elbow. There are fits of dyspnoea, even when she is at rest. Apex-pulsation is now felt one inch outside the nipple-line, and here, as previously, a loud postdiastolic murmur of blowing quality is heard. To have carbonate of ammonia, grs. iij, with tincture of digitalis and chloric ether, of each ℥v, thrice daily; grs. v of Dover's powder at night, and xxx drops of ozonic ether (Richardson) by inhalation, during paroxysms of dyspnoea.

November 24th. Has repeatedly spat blood since last report. Cough is now very troublesome; severe pain in region of heart; acoustic signs as previously noted. To have two leeches at seat of pain, and a cough mixture containing chlorodyne and morphia.

February 2nd, 1870. A fortnight since she threw up a large quantity of blood, after which she fainted, and was insensible for about an hour. One week later, the hæmoptysis and syncope recurred whilst she was at work, and on that occasion, according to the report of her companions, her face became congested. Pulse 110, and regular; feet somewhat swollen in the afternoon; cheeks congested and purple; pain at apex of heart, and orthopnoea. The cardiac signs were unaltered.

She was admitted into hospital on the 3rd of February, and remained under treatment about two months. During this period she coughed up large quantities of blood from time to time, and suffered from severe pain at the heart. The latter was always promptly relieved by leeching. The annexed tracing (Fig. LXI.) represents the character of the pulse at that date.

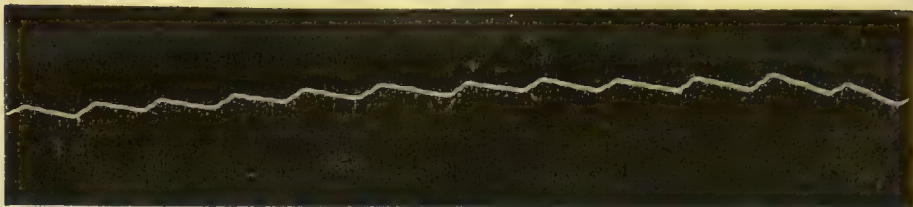


FIG. LXI.

Extreme mitral constriction.

Mary E. C. Pulse regular, but very feeble. March 24th, 1870.

October 31st. Several weeks ago, whilst at work, she was struck on the left side of the abdomen with great violence, by the handle of a machine, and was rendered incapable of resuming her labour for some time. Kidneys acting defectively; cough and dyspnoea; feet swollen at night. There has been occasionally a streak of blood in the sputa; she complains of pain at the seat of injury, and likewise at the precordium. At the point of apex-pulsation, which is now located one inch and a-half externally to the nipple-line, a faint presystolic, and a loud postdiastolic, murmur are audible; both murmurs may be likewise heard, but much less distinctly, midway between the nipple-line and the sternum; they are not audible at the base, where the sounds of the heart are normal. Pulse 96, regular, and moderately full. To have tincture of digitalis and chloric ether, of each ℥v, thrice daily.

October 19th, 1871. After an absence of nearly a year, this girl called on me to-day. She now suffers occasionally at night from pain of a burning character in both scapular regions, but more severe on the right than on the left side. Pulse 108, regular, and moderately strong; dyspnoea; congestion of face, and œdema of the ankles after the exertion of the day; dysuria. There is scarcely any cough, but the sputa are occasionally bloody. She can now sleep, and can pursue her avocation (machine sewing), but with some fatigue.

The apex-beat was one inch and a-half to the left of the nipple-line, and in this situation a postdiastolic murmur was alone heard. Cardiac sounds normal, and elsewhere unattended with murmur. To have tincture of digitalis with spirit of nitrous ether; two leeches to be applied in left infra-scapular region.

February 5th, 1872. Has had a febrile attack which confined her to bed for six weeks; no hæmoptysis for the last four months; great dyspnoea on ascending a flight of stairs, and likewise when she attempts to work the treadle of her machine; but after a short time the difficulty of breathing subsides, and she can then continue her work. Feet still swollen at night; face purple, and eyes prominent; congestion of right lung; pulse 84, small and regular. Apex-beat in fifth interspace, one inch and a-half outside the nipple-line, and felt over an area of one inch in diameter. In this situation, but strictly limited to it, a loud postdiastolic murmur is audible; it is continued from, but in no degree masks, the second sound, whilst it falls short of the first sound by a distinct interval of time. Both cardiac sounds are normal. The second sound is accentuated in the pulmonary artery.

April 29th. For last fortnight she has been suffering from irritability of stomach, and empty retching; has a feeling of weight at the pit of the stomach, and a "burning" sensation in both scapular regions. To have mild aperients, and Boudault's pepsine.

February 15th, 1873. Admitted again into hospital to-day. Is very weak; has cough and headache; face of a bright crimson tint; pulse 96, regular; no œdema. Apex-pulsation two inches external to nipple-line, and here there is a harsh and prolonged postdiastolic murmur, which is accompanied by fremitus; this is succeeded by a short period of silence; and then a harsh presystolic murmur and a normal first sound are heard in succession. No murmur is elsewhere audible. The second sound is not double or accentuated. On one occasion, whilst in hospital, she coughed up a teacupful of florid blood. She likewise complained of pain at the apex of the heart, which was relieved by the application of two leeches, followed by the administration of ℥xxx doses of the tincture, with 3j of the syrup, of the American wild cherry (*Prunus Virginiana*). The pulse, at this date, is represented in the tracing (Fig. LXII.).

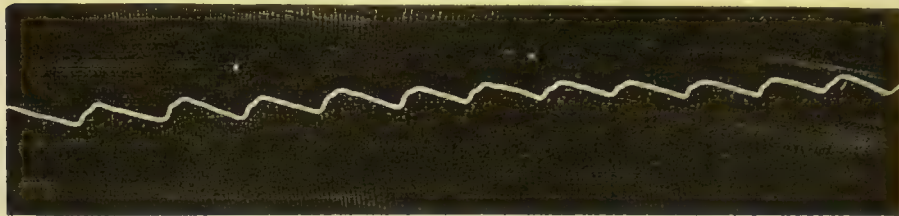


FIG. LXII.

Mitral constriction. Mary E. C. June, 1873.

August 15th, 1874. Called on me to-day. Complains of pain beneath the left nipple, and in left back; has pain also in the joints of feet and hands. The face is purple; the breathing oppressed; she has a dry and teasing cough. Pulse 96, and regular. A loud and "harsh" postdiastolic murmur only exists at the apex of the heart.

The protracted period, more than six years, during which this case has been under my observation, and the great interest which attaches to it in regard to the progressive development of physical signs, may be accepted as my apology for publishing it, and at such length, in connexion with those in which the diagnosis was confirmed by autopsy.

CASE CXVII.—*Rheumatism; Dissipation; Hæmoptysis; Dyspœna and Œdema; Hepatic Congestion; Double Cardiac Impulse; Fremitus; Postdiastolic and Presystolic Murmur at Apex; Postdiastolic Murmur with Musical Note, at Base; Partial Left Hemiplegia and Left Hemiopia; Extensive Sub-retinal Extravasation. Diagnosis: Mitral Stenosis; Inadequacy of Aortic Valve from Laceration of one or more of the Segments; Capillary Embolism.*

Thomas K., aged twenty-four years, a man of very intemperate habits, and a float-driver by occupation, was received into hospital on the 16th of September, 1874. He had been previously admitted for rheumatic pains, from which he quickly recovered without having apparently sustained any serious detriment of health.

He had been recently leading a very irregular life; had drunk



a good deal of whiskey, and been exposed to hardship of various kinds. For nine days before admission, he had been expectorating florid blood, and for several nights he had not been able to lie down, owing to the difficulty of breathing which he experienced in the horizontal posture. When admitted, he was pale, bloated-looking, and very weak; he complained of a feeling of constriction at the sternum, and breathed with great difficulty; the feet were swollen. From a cursory examination made in the waiting room, I diagnosed mitral constriction and aortic regurgitation.

The man was placed under the care of Dr. Nixon, who, at my request, kindly transferred him to me on the following day.

The report on the 18th was as follows: He suffers from teasing cough with expectoration of blood-stained mucus; there is orthopnoea, also cedema of the feet, visible pulsation of the carotids, and epigastric tenderness; pulse 96, full and abrupt, but not visible. The impulse of the heart was double. Apex-pulsation was felt half an inch inside the nipple-line, in the fifth interspace; at this point, in addition to the sounds of the heart, which were distinct and normal, two murmurs were audible; they were respectively postdiastolic and presystolic in rhythm. At the base, both sounds of the heart were sharp and abrupt, and the second was succeeded by a soft bellows-murmur which was accompanied by a musical note. This murmur was transmitted with great distinctness to the ensiform cartilage, and faintly to the right second costal cartilage, but not into the transverse portion of the arch, or the carotid arteries. No murmur was audible in the back. The base of the right lung was congested. The slightest movement of the body was followed by great palpitation and respiratory distress.

Leeches were applied at midsternum, the chest was dry-cupped posteriorly, and a mixture of chloric ether and tincture of digitalis was given. Fig. LXIII. shows the character of the pulse on the following day.

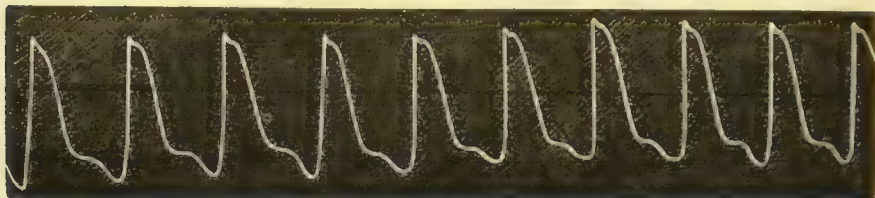


FIG. LXIII.

Mitral constriction and partial aortic regurgitation. Hypertrophy and dilatation of the left ventricle.

Thomas K. Pulse 112. September 19th, 1874.

On the night of the 20th he was suddenly attacked with headache, and with severe pain in the left side of the chest, extending down the left arm and leg. On the following morning he had lost vision in nearly one-half of the left retina; he could only see the upper portion of objects with that eye. The pupils were, however, normal and equal; hæmoptysis had continued. Fremitus was now detected at the apex, and in that situation only a single murmur was heard, which, however, extended over the entire long pause.

Three leeches were applied over the apex of heart, and gr. ij each of grey and of antimonial powder were given every second hour.

On the 23rd he was much quieter, having slept well the preceding night. The apex-murmur was now again double, *i. e.*, it consisted of a postdiastolic and a presystolic element, with an intervening period of silence; the latter was ingravescent; it was also more harsh than the former. The first sound at the base was accompanied by a faint murmur which was transmitted into the carotid arteries; whilst the second sound was accentuated in the aorta, and its satellite murmur was now audible in the transverse portion of the arch. The left eye underwent a careful examination with the ophthalmoscope to-day, by my colleagues, Mr. Hayes and Dr. Nixon. An extensive extravasation of blood was discovered in the fundus, by which the upper and outer portion of the retina was raised in large and irregular patches, radiating from the optic entrance.

The man can now sleep in the recumbent posture, is free from pain in the arm and leg, and from feeling of constriction in the chest. The œdema has been dispersed.

On the 28th he was allowed to be out of bed. For the preceding few days he had been taking, thrice daily, a draught

consisting of Hoffman's anodyne, 3ss; Tincture of camphor and spirit of chloroform, of each, ℥xv. From this he appeared to derive great benefit.

There can, I think, be little doubt that capillary embolism, by migration of detritus from the aortic valve, took place on the night of the 20th, and was the cause of the pain in the left side of the body, and of the partial loss of vision by subretinal hæmorrhage in the left eye.

In cases of hemiplegia from embolism of the middle cerebral artery, so frequently witnessed in connexion with valvular disease on the left side of the heart, I entirely agree with Dr. Clifford Allbutt, and therefore differ from Niemeyer, in holding that loss of consciousness is of very rare occurrence. There is usually nothing more than temporary bewilderment from shock. The order of events which may be witnessed in cases of embolism of the central artery of the retina, is, according to Allbutt, the following, namely; instantaneous loss of function, sudden emptying of retinal vessels, gradual œdema of portions of retina deprived of blood, hæmorrhage still later from collateral vessels, thickening of "adventitia," and fatty degeneration of retina with deposit of chlosterine.\*

The subjoined tracing, though representing a combination of valvular disease identical as to kind with that shown in the preceding figure (LXIII.), contrasts strikingly with the latter by its want of regularity, and by the defective height and verticality of its upstroke. It represents, in short, incipient failure of the left ventricle from retrogressive tissue change.

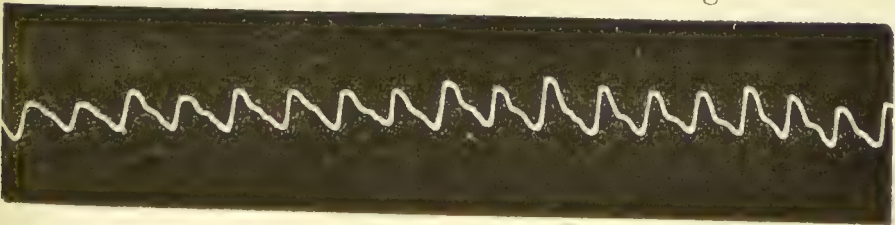


FIG. LXIV.

Mitral obstruction, and partial aortic regurgitation.

Peter F. (No. 61, Table XI.) September 23rd, 1873.

All the cases of mitral stenosis which came under my notice previously to 1875, are presented, in summary, in the succeeding Tables (XI. and XII.).

\* *On the Use of the Ophthalmoscope*, 1871.

TABLE XI.—MITRAL STENOSIS.

No.	Name.	Age.	Hemoptysis.	Dyspnea.	Edema.	Irregular or Intermittent Pulse.	Failure of Pulse.	Frenitulus.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
1	Anne K. ..	28	1	1	1	1	1	1	..	1	..	1	1	Tricuspid inadequacy, vegetations on valve.	Funnel-shaped.	..
2	Ellen D. ..	30	..	1	1	1	..	1	1	1	..	..	..	..	..	..
3	Jane L. ..	33	1	1	1	1	1	1	1	1	..	..	1	..	..	..
4	Jane G. ..	34	1	1	1	1	1	1	1	1	..	..	1	..	..	..
5	Jane Q. ..	47	1	1	1	1	1	1	1	1	1	1	1	..	..	..
6	Mary S. ..	26	1	1	1	1	1	1	1	1	1	1	1	..	..	..
7	Jane McG... ..	50	1	1	1	1	1	1	1	1	1	1	1	..	..	..
8	John M. ..	*	1	1	1	1	1	1	1	1	1	1	1	..	..	..
9	John M. ..	50	1	1	1	1	1	1	1	1	1	1	1	..	..	..
10	Agnes B. ..	48	1	1	1	1	1	1	1	1	1	1	1	..	..	..
11	Mary M. ..	37	1	1	1	1	1	1	1	1	1	1	1	..	..	..
12	Robert McB. ..	14	1	1	1	1	1	1	1	1	1	1	1	..	..	..
13	Thomas N. ..	10	1	1	1	1	1	1	1	1	1	1	1	..	..	..
14	Mary F. ..	30	1	..	..	..	..	1	..	1	1	1	1	..	..	..
15	Mary B. ..	28	..	1	1	1	..	1	1	..	..	..	..	Double aortic.	..	..
16	Patrick C. ..	40	1	1	1	1	..	1	1	1	1	1	1	..	..	..
17	Kate O'B. ..	24	1	1	1	1	..	1	1	1	1	1	1	..	..	..
18	Margaret B. ..	30	..	1	1	1	..	1	1	1	1	1	1	..	..	..
19	Patrick Q. ..	40	1	1	1	1	..	1	1	1	1	1	1	..	..	..
20	Bridget P. ..	24	1	1	1	1	..	1	1	1	1	1	1	Double aortic.	..	..
21	Charles B. ..	17	1	1	1	1	..	1	1	1	1	1	1	..	..	..
22	Sarah M. ..	26	1	1	1	1	..	1	1	1	1	1	1	Tricuspid and aortic obstruction.	Button-hole.	..
23	Bridget McG. ..	23	1	1	1	1	..	1	1	1	1	1	1	Aortic insufficiency.	..	..
24	Mrs. E. ..	38	..	..	..	1	..	1	1	1	..	..	..	Aortic insufficiency.	..	..



No.	Name.	Age.	Hemoptysis.	Dyspnea.	Edema.	Irregular or Inter- mittent Pulse.	Failure of Pulse.	Fremitus.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
25	Lizzie H. ..	18	1	1	1	1	1	1	1	1	1	1	1	Aortic insufficiency.	..	..
26	Eliza H. ..	26	1	1	1	1	1	1	1	1	1	1	1	..	..	..
27	Mary O'S.	22	..	..	..	..	..	..	..	..	..	..	..	..	..	..
28	Anne R. ..	23	..	..	..	..	..	..	..	..	..	..	..	..	..	..
29	Sarah C. ..	46	1	..	..	..	..	..	1	..	..	..	..	..	..	..
30	John M. ..	*	..	..	..	..	..	..	1	1	..	..	..	..	..	..
31	John C. ..	22	..	..	..	..	..	..	1	1	..	..	..	..	..	..
32	Patrick McD.	23	1	1	1	1	..	1	1	..	..	1	1	Aortic obstruction and inadequacy; and tricuspid ob- struction and in- adequacy.	Button-hole.	Embolism and left hemiplegia. ..
33	Mrs. R. ..	30	..	..	..	..	..	1	..	..	..	..	..	..	..	..
34	Joseph F. ..	*	..	..	..	..	..	1	..	..	..	..	..	..	..	..
35	Mary M. ..	7	..	1	..	..	..	1	..	..	..	..	..	..	..	..
36	Miss C. ..	38	..	1	..	..	..	..	1	..	..	..	..	..	..	..
37	Miss H. ..	38	1	1	1	..	..	..	..	..	..	..	..	Aortic obstruction and inadequacy.	..	..
38	Mrs. R. ..	25	1	..	..	..	..	..	..	1	..	..	..	..	..	First sound of heart double.
39	Thomas D.	15	..	1	..	..	..	1	1	..	1	..	..	..	..	..
40	Emma McC.	*	..	..	..	..	..	1	1	..	..	..	..	..	..	..
41	Mary K. ..	26	..	..	..	..	..	1	..	..	..	..	..	..	..	..
42	Jane C. ..	24	..	1	..	..	..	1	..	1	..	..	..	..	..	..
43	Thomas M.	16	1	..	..	..	..	1	1	1	..	..	..	..	..	..
44	Robert F.	7	..	1	..	..	..	1	..	..	..	1	1	Aortic obstruction and inadequacy.	Button-hole.	Embolism, right hemiplegia, and aphasia.
45	Mary A. ..	28	..	1	1	..	1	..	1	..	1	1	1	Aortic obstruction.	Button-hole.	Fatty degeneration of heart (see case 61, p. 710).

\* Age not stated.

TABLE XI.—*Concluded.*

No.	Name.	Age.	Hemoptysis.	Dyspnea.	Edema.	Irregular or Inter- mittent Pulse.	Failure of Pulse.	Pneumitis.	Rheumatism.	Doubled 2nd Sound.	Accent. 2nd Sound in P. Artery.	Systolic Murmur.	Autopsy.	Other Valve-lesion.	Form of Mitral Stenosis.	Complications and Observations.
46	Kate B. ..	18	..	..	..	..	..	1	1	1	1	..	..	..	..	Had five different attacks of rheumatic fever.
47	Bridget McG. ..	28	1	..	..	..	..	1	1	1	1	1	..	..	..	Had three attacks of rheumatism; pre-systolic murmur became inaudible in state of debility.
48	Mary F. ..	23	..	..	..	1	1	1	..	1	..	..	..	..	..	Embolism; right hemiplegia and aphasia.
49	Michael C. ..	23	1	1	..	..	..	..	1	..	..	..	1	Aortic obstruction and inadequacy.	Button-hole.	Right hemiplegia & partial aphasia; clot in left cerebral hemisphere.
50	Mary K. ..	26	..	..	..	..	..	..	..	..	..	..	..	..	..	Embolism; right hemiplegia and aphasia; recovery.
51	Margaret C. ..	30	..	..	..	..	..	..	..	..	..	..	..	..	..	..
52	Michael F. ..	25	1	..	..	1	1	1	..	..	..	..	..	Tricuspid obstruction.	..	..
53	Mary McC.	40	1	1	1	..	..	..	..	..	..	..	1	Aortic obstruction.	Button-hole.	..
54	Thomas W.	36	1	1	1	..	..	..	..	..	..	..	..	Aortic inadequacy.	..	..
55	Patrick McD.	52	1	1	1	..	..	..	..	..	..	..	..	..	..	..
56	Daniel G. ..	27	..	..	..	..	..	..	..	1	..	..	..	..	..	..
57	Michael L.	17	1	..	..	..	..	..	..	..	..	..	..	..	..	..
58	Theresa J.	12	..	..	..	..	..	..	..	1	1	1	..	..	..	..
59	Elizabeth B.	21	1	..	..	..	..	1	1	1	..	..	..	..	..	..
60	Bessie L.	50	1	..	..	..	..	..	..	1	..	..	..	..	..	..
61	Peter F.	48	1	..	..	..	..	..	..	1	..	..	..	..	..	..
62	Eliza E.	27	..	..	..	..	..	1	..	1	..	..	..	..	..	..
63	Eliza D. ..	17	..	..	..	..	..	1	1	..	1	1	..	..	..	..
63			35	24	17	14	9	34	31	26	15	18	13			

TABLE XII.—MITRAL STENOSIS (SUPPLEMENTARY).

No.	Name.	Age.	Hæmoptysis.	Dyspnea.	Edema.	Irregular and Inter- mittent Pulse.	Failure of Pulse.	Feveritus.	Rheumatism.	Double 2nd Sound.	Accent. 2nd Sound.	Systolic Murmur at Aper.	Autopsy.	Other valves affected.	Form of Mitral Stenosis.	Complications and Observations.
1	Mr. B.	42	..	..	..	..	..	..	..	1	..	..	..	..	..	Sudden amnesia of words, from which she re- covered.
2	Catherine P.	43	..	..	..	..	..	..	..	..	..	..	..	..	..	Neuralgia.
3	Mrs. McH.	40	..	..	..	..	..	..	..	..	..	..	..	..	..	Leg amputated for bad sore, by Mr. Hayes; pa- tient made a good recovery.
4	Eliza C.	50	1	..	..	..	..	..	..	..	..	..	1	Tricus- pid ste- nosis.	Funnel- shaped in both.	Cold after measles in infancy.
5	Margaret M.	25	1	1	1	1	1	..	..	..	..	..	..	..	..	Acute pericarditis.
6	Arthur C.	14	..	1	..	..	..	1	1	1	1	1	..	..	..	Acute pericarditis; loud metallic tinkle in sto- mach synchronous with impulse of heart.
7	Thomas H.	25	..	..	..	..	..	1	1	1	1	1	..	..	..	Presystolic murmur was suppressed for two days; after which it was again audible, and continued to be heard until she was discharged.
8	Margaret McG.	25	1	..	..	..	..	..	*	1	..	..	..	Aortic inade- quacy.	..	Had seven different attacks of rheumatism. Three and a-half years previously I diagnosed mitral stenosis. Murmur inaudible for a period of two days. Localized pericardial friction-sound of systolic rhythm, at base.
9	Essie McK.	19	..	..	..	..	..	..	1	..	..	1	..	..	..	Had three several attacks of rheumatism. Pulmonary emphysema and displacement of heart down, behind ensiform cartilage, where presys- tolic murmur was heard; it was distinguished from first element of a double first sound, by absence of coincident impulse, by its preceding carotid pulse, and, especially, by its harsh quality.
10	Bridget G.	27	..	1	..	..	..	1	..	..	..	1	..	..	..	..
11	John McG.	53	..	..	..	..	..	..	..	..	..	..	..	..	..	..

\* Not stated.

TABLE XII.—SUPPLEMENTARY.—*Concluded.*

No.	Name.	Age.	Hæmoptysis.	Dyspnoea.	Œdema.	Irregular and Intermittent Pulse.	Failure of Pulse.	Fremitus.	Rheumatism.	Double 2nd Sound.	Accent. 2nd Sound.	Systolic Murmur at Apex.	Autopsy.	Other valves affected.	Form of Mitral Stenosis.	Complications and Observations.
12	Eliza B.	24	1	1	1	..	..	1	1	..	1 (in aorta)	..	..	..	..	Sister to Kate B., No. 46, Table XI. Inadequacy of aortic valve, most probably by rupture of one or more of the segments. Partial left hemiplegia and left hemiopia, by embolism; hæmorrhage beneath left retina. Albuminuria and consecutive hypertrophy of the heart; endo-myocarditis, and systolic murmur. (See case 68.) Face occasionally livid, and swollen in the morning. For last month has been cyanosed. Pulmonary emphysema. General hypertrophy of the heart; albuminuria and hæmaturia; no presystolic murmur during the thirty-nine days of patient's residence in hospital.  Recurrent hæmoptysis; cyanosis; exophthalmia; syncope; presystolic murmur occasionally heard; and persistent postdiastolic murmur. Double impulse; increased area of precordial dulness; acute renal congestion; hæmaturia with albuminuria.
13	Thomas K.	24	1	1	1	..	..	1	1	..	1	..	..	Aortic inadequacy.	..	
14	Mrs. B.	30	1	1	1	..	..	..	..	1	1	1	..	Mitral inadequacy.	..	
15	Mary E.	56	1	1	1	..	1	..	..	..	..	..	..	Tricuspid stenosis (?)	..	
16	Matthew N.	58	1	1	1	..	..	..	1	..	..	1	1	Mitral and Tricuspid inadequacy.	Button-hole.	
17	Mary E. C.	25	1	1	1	..	..	1	1	..	1	..	..	..	..	
18	Michael O'B.	21	1	1	1	..	..	1	1	1	..	1	..	Mitral inadequacy.	..	
18			9	9	7	..	2	6	9*	4	5†	7	2			

\* Not stated, 2.

† 3 in pulmonary artery, 2 in aorta.



It appears to me that some additional light may be thrown upon the statistics of mitral stenosis, and upon the relative frequency of its various complications, by a careful study and comparison of the results obtained from Dr. Hilton Fagge's cases, and my own as epitomised in Tables XI. and XII. For the calculations in both instances I am alone responsible. I have made them with the greatest care, and I hope they may be found at least substantially accurate.

I may here briefly state that Dr. Fagge's cases, 66 in number, are grouped under three heads, namely:

1. Those in which a presystolic murmur was heard during life, and the mitral orifice was found, after death, to be contracted; these cases were 7 in number.

2. Those in which the mitral orifice was found, on *post mortem* examination, to be contracted, but in which presystolic murmur had not been heard during life; the number of such cases was 40.

3. Cases in which a presystolic murmur was identified, but no opportunity was afforded for testing the diagnosis by *post mortem* examination; these cases numbered 19.

The fatal cases in this list were, therefore, 47 in number. A diagnosis was made in 7 instances, and verified by the result.

The total of my cases was 81. Out of this number 16 deaths took place, and the body was examined in 15 instances. The diagnosis of mitral narrowing was not made in 3 of the last-mentioned 15 cases, owing to the absence of presystolic murmur during the time the patients had been under observation. It was made in the 12 remaining cases, and in all it was verified by the result.

TABLE XIII.—ABSTRACT OF CASES OF MITRAL STENOSIS.

DR. FAGGE.		DR. HAYDEN.	
<i>Ages of Patients.</i>		<i>Ages of Patients.</i>	
Total cases, 66.	{ Under 10 years ... 0	Total cases, 81.	{ Under 10 years ... 2
	10 and under 15 years 8		10 and under 15 years 4
	15 " 20 " 5		15 " 20 " 8
	20 " 25 " 7		20 " 25 " 14
	25 " 30 " 8		25 " 30 " 18
	30 " 35 " 8		30 " 35 " 8
	35 " 40 " 1		35 " 40 " 5
	40 " 45 " 9		40 " 45 " 6
	45 " 50 " 8		45 " 50 " 4
	50 " 55 " 4		50 " 55 " 6
Average age of 61 patients = 32.77.	55 " 60 " 1	Average age of 77 patients = 29.63.	55 " 60 " 2
	60 and over ... 2		60 and over ... 0
	Age not stated ... 5		Age not stated ... 4
	— 66		— 81
Fatal cases in which the body was examined, 47.	{ Under 10 years ... 0	Fatal cases in which the body was examined, 15.	{ Under 10 years ... 1
	10 and under 15 years 2		10 and under 15 years 2
	15 " 20 " 1		15 " 20 " 0
	20 " 25 " 5		20 " 25 " 2
	25 " 30 " 5		25 " 30 " 4
	30 " 35 " 5		30 " 35 " 2
	35 " 40 " 1		35 " 40 " 1
	40 " 45 " 9		40 " 45 " 0
	45 " 50 " 7		45 " 50 " 1
	50 " 55 " 4		50 " 55 " 1
Average age of 42 patients = 37.83.	55 " 60 " 1	Average age of 15 patients = 29.26.	55 " 60 " 1
	60 and over ... 2		60 and over ... 0
	Age not stated ... 5		Age not stated ... 15
	— 47		— 15
<i>Sex of Patients.</i>		<i>Sex of Patients.</i>	
Males, Females, Sex not stated, ...	28 in 65 cases	Males, Females, Sex not stated, ...	27 in 81 cases
	37 in 65 "		54 in 81 "
	1		==
66		33.3 per cent. 66.7 "	

*History of Symptoms.*

Antecedent Rheumatism	...	33 in 66 cases	=	50.0 per cent.
Hæmoptysis	...	3 in 66 "	=	4.5 "
Irregularity of Pulse	...	16 in 66 "	=	24.2 "
Reduplication of Second Sound	...	3 in 66 "	=	4.5 "

*Complications.*

(	Aortic obstruction and inadequacy	...	4	} 24 in 66 cases = 36.3 per cent.
	Aortic obstruction & tricuspid inadequacy	...	2	
	Aortic obstruction only	...	1	
	Tricuspid inadequacy	...	1	
	Mitral inadequacy and aortic obstruction and inadequacy	...	2	
Other valve lesions.	Mitral inadequacy	...	14	
	Mitral and tricuspid	...	1	

Pulmonary apoplexy

6 in 47 cases = 12.7 per cent.

Hemiplegia from embolism in total cases.

Right, with aphasia	1
Right, without aphasia	1
Left, with aphasia	2
Left, without aphasia	1
Side not stated	1

5 in 66 cases = 7.5 per cent.

Hemiplegia from embolism in fatal cases.

4 in 47 cases = 8.5 per cent.

Embolism of lower limb

3 in 66 cases = 4.5 per cent.

Figure of contracted mitral orifice

46 in 47 cases = 97.8 per cent.

Funnel-shaped

1 in 47 cases = 2.1 per cent.

*History of Symptoms.*

Antecedent Rheumatism	...	40 in 81 cases	=	49.3 per cent.
Hæmoptysis	...	44 in 81 "	=	54.3 "
Irregularity of Pulse	...	15 in 81 "	=	18.5 "
Reduplication of Second Sound	...	30 in 81 "	=	37.0 "

*Complications.*

(	Aortic obstruction, ...	2	} 47 in 81 cases = 58.0 per cent.
	Aortic inadequacy ...	10	
	Aortic obstruction and inadequacy	4	
	Tricuspid inadequacy	1	
	Tricuspid obstruction	4	
Other valve lesions.	Tricuspid and aortic obstruction and inadequacy, with mitral inadequacy	1	
	Mitral inadequacy	24	
	Mitral and tricuspid inadequacy	1	

Pulmonary apoplexy

8 in 15 cases = 53.3 per cent.

Hemiplegia from embolism in total cases.

Right, with aphasia	5
Right, without aphasia	0
Left, with aphasia	0
Left, without aphasia	2

7 in 81 cases = 8.6 per cent.

Hemiplegia from embolism in fatal cases.

3 in 15 cases = 20.0 per cent.

Figure of contracted mitral opening.

Button-hole

13 in 15 cases = 86.6 per cent.

Funnel-shaped

2 in 15 cases = 13.3 per cent.

In addition to those given in the preceding list, at least fifteen examples of mitral stenosis, accompanied by full details of *post mortem* examination, have been recorded in Ireland since 1855. In some of these cases a positive, and in others a presumptive, diagnosis was made.

The names of the authors, and the references to the record of the cases, arranged chronologically, are briefly as follows :

- DR. M'DOWEL : mitral stenosis ; no murmur audible ; diagnosis made from general symptoms. (*Proceedings of the Dublin Pathological Society.*) December 1st, 1855.
- DR. BANKS : aortic and mitral narrowing. (*Ibid.*) December 6th, 1856.
- DR. WILLIAM MOORE : mitral stenosis ; diagnosis of mitral disease. (*Ibid.* vol. i., part ii., p. 80.) March 24th, 1860.
- DR. LEES : mitral obstruction ; general diagnosis. (*Ibid.*, vol. i., part ii., p. 84.) April 14th, 1860.
- DR. STOKES : mitral, aortic, and tricuspid obstruction ; diagnosis of mitral narrowing. (*Ibid.*, vol. ii., part i., p. 14.) December 6th, 1862. (Case referred to at p. 1011.)
- DR. HEAD : mitral stenosis. (*Ibid.*, vol. ii., part iii., p. 165.) November 26th, 1864.
- DR. DUNCAN : mitral stenosis. (*Ibid.*, vol. ii., part iii., p. 186.) March 26th, 1865.
- DR. MACSWINEY : mitral stenosis. (*Ibid.*, vol. iii., part i., p. 132.) March 3rd, 1867.
- DR. HAWTREY BENSON : mitral and aortic obstruction, both diagnosed ; apex presystolic murmur identified. (*Ibid.*, vol. iv., part i., p. 35.) April 24th, 1869.
- DR. CRYAN : mitral and tricuspid obstruction ; diagnosis of double lesion ; presystolic murmur identified. (*Ibid.*, vol. iv., part ii., p. 144.) February 12th, 1870. (Case epitomised, p. 1013.)
- DR. MAGRATH (8th Hussars) : mitral stenosis. (*Ibid.* vol. iv., part iii., p. 250.) January, 1871.
- DR. CRYAN : mitral and tricuspid obstruction ; presumptive diagnosis of mitral stenosis from general symptoms ; no presystolic murmur audible. (*Ibid.*, vol. v., part i., p. 17.) April 20th, 1872. (Case epitomised, p. 899.)
- DR. GERALD YEO : extreme mitral constriction ; aortic obstruction and incompetency ; hypertrophy of the right ventricle ; hæmorrhagic infarction of the lungs ; and extensive atheroma of the branches of the pulmonary artery in both lungs ; diagnosis of the twofold valve-lesion. (*Ibid.*, vol. iv., part ii., p. 123.) December 14th, 1872.
- DR. NIXON : mitral obstruction and aortic patency ; both diagnosed ; presystolic murmur recognized. (*Ibid.*, vol. v., part ii., p. 133.) February 8th, 1873. (See case 112.)
- DR. JOHN W. MARTIN, of Portlaw : mitral and aortic obstruction ; both diagnosed ; presystolic murmur recognized. (*Medical Press and Circular.*) March 11th, 1874.

I cannot bring my remarks on this subject to a conclusion without observing that, in 1827, Mr. Adams of Dublin antici-



pated most of what has been since done in regard to the diagnosis of mitral narrowing.\*

That he recognised, at least in outline, the distinctive signs of this lesion, will appear from the following extract from his well-known memoir.† “When the ear is attentively applied to the side of the thorax, a very complex kind of movement, hard to describe, is heard. A hissing, purring noise, as it has been denominated, caused by the transmission of blood through a narrow orifice, is in most cases very evident.” He adverts to the dilatation and hypertrophy of the left auricle and right ventricle, and to the pulsation of the jugular veins, synchronous with that of the heart, and due to tricuspid reflux from inadequacy of the valves.

The fremitus and reduplicated second sound were described in the following words: “That the organ (heart) performed some kind of double movement, and that its motions were accompanied with a purring sound, as in varicose aneurism, was plainly audible, and also could be recognized when the hand was placed over the region of the heart.” His failure to appreciate the presystolic rhythm of the murmur and fremitus constitutes the only shortcoming in this graphic sketch. Thus, it would seem that, after Bertin, Dr. Adams was the first physician who identified mitral narrowing by its peculiar physical signs.

*Inadequacy of the mitral valve* from structural alteration of one or both of its segments, constitutes the most common form of valvular lesion.‡ This is due to the circumstance that the mitral valve is liable, not only to disease and injury proper to itself, but likewise to the consequences of retrograde pressure from disease at the orifice of the aorta.

The mitral valve may, however, be incompetent without having undergone any change of structure whatever. Cases belonging to the latter category may be reduced under the following heads: (a) Valvular inadequacy and regurgitation from displacement of one or both segments of the valve, through the papillary muscles and tendinous chords, by excentric yielding of the walls of the left ventricle at the acme of systole. This result

\* This eminent man has just departed from amongst us (January, 1875).

† *Dublin Hospital Reports*, vol. iv.

‡ See p. 815.

may be due to fatty degeneration of the left ventricle, as exemplified in Case 49, p. 685; and in Case 50, p. 687. The remarks upon this subject at pages 282, 558, 562, and 690, may be referred to as explanatory of my view of the pathology of reflux from this cause. It may be likewise attributable to debility, without structural alteration of the valves or of the ventricle, from anæmia, malnutrition, or asthenic disease.\* (b) Regurgitation from rupture of one or more of the primary tendinous chords.† (c) From dilatation of the left ventricle without proportionate elongation of the papillary muscles.‡ (d) Weakness and relaxation of the papillary muscles.§ (e) Adhesion of the tendinous chords to the walls of the ventricle.|| (f) Engagement in the orifice, of a pedunculated mass of fibrin, depending from its attachment to one of the curtains of the valve, or to the interior of the auricle.¶ (g) Fixation of the anterior segment of the mitral valve by a calcareous mass descending from the aortic orifice, and attached to the valve.\*\* For the reasons already given, I cannot admit, as a cause of mitral reflux, spasm or irregular action of the papillary muscles;†† nor can I recognize murmur from excessive tension of the valves, without regurgitation.‡‡

In the great majority of cases, inadequacy of the mitral valve arises from antecedent inflammation and morbid alteration of its structure. Rheumatic endocarditis is the ordinary cause of the change just referred to. Inflammatory irritation consecutive to disease of the aortic valves, may lead to the same result. This is the cause to which active disorganization of the mitral valve,

\* See pages 275, 282; Case 96, p. 881; also a case by Dr. Grimshaw, *Proceedings of the Pathological Society of Dublin*, vol. vi., part i., p. 18.

† See p. 235; Case 24, p. 535; also a case by Dr. Gordon, *Stokes on Diseases of the Heart*, Case 68, p. 473; and a case by Dr. A. W. Foot, *Proceedings of the Pathological Society of Dublin*, vol. vi., part i., p. 15.

‡ See p. 278-9.

§ See p. 280.

|| Niemeyer, *Opus citat.*, vol. i., p. 352.

¶ Walse, *Diseases of the Heart*, fourth edition, p. 93. An example of this kind was exhibited to the Pathological Society, by Dr. R. McDonnell, in Dec., 1871.

\*\* Bristowe, *Transactions of the Pathological Society of London*, vol. iii., p. 157.

†† See p. 276.

‡‡ See p. 280.

of non-rheumatic origin, is in most cases to be attributed; the exceptions would include examples of uræmic, septicæmic, and traumatic valvulitis.

Inadequacy of the aortic valves, by the increased strain upon the walls of the left ventricle to which it gives rise, determines, at once, dilated hypertrophy of that chamber, and thickening by interstitial growth, of the mitral valves. In consequence of the structural changes which the valves have thus undergone, they are incapable of expanding proportionately to the further increase in the capacity of the ventricle, and mitral regurgitation is the necessary result, as Dr. Milner Fothergill states.\* That in such cases, dilatation of the left ventricle is the principal factor of the mitral inadequacy, is shown by the absence of the latter lesion in cases of simple hypertrophy from renal disease, or from functional excitement.

Doctor Fothergill holds that in such cases the mitral valves are closed, and the strain upon them continued during ventricular diastole, by the reaction of the aorta. I am not satisfied upon this point. I apprehend that if such were the case, a diastolic murmur of mitral origin should be an invariable precursor of consecutive mitral inadequacy; but that it is not so related to the latter lesion is well known.

The pathology and symptoms of acute endocarditis, to whatever cause due, have been already discussed.† The mitral valve, rendered incompetent by inflammatory changes, is always thick and opaque. Whilst still affected by acute inflammation, the segments are swollen, but even on the surface, and of a uniform crimson hue; at a later stage they are of a dull white colour, more or less irregular on the surface, and firm in texture. The subsequent changes of the ordinary character consist in shrinking and retraction, with increased thickening and rigidity of the valve-segments.

The density of structure may attain to cartilaginous hardness, and in gouty subjects deposition of lime-salts and calcareous change may ultimately ensue. In this latter contingency, the surface and edges of the valves are rendered rough and spicu-

\* *Lancet*, May 16th, 1874.

† See pp. 802-6.

lated, and so rigid, that obstruction usually coincides with inadequacy of the valves. The changes above mentioned, except calcification, may extend to the tendinous chords.

Inadequacy of the mitral valve necessarily involves systolic reflux upon the left auricle. The contractile force of the left ventricle is thus expended in part upon that chamber, which soon exhibits the effects of increased pressure in the consequent changes of dilatation and hypertrophy. During ventricular systole the left auricle is slowly filled, and within the measure of its normal capacity, by influx from the pulmonary veins only; but when the mitral valve is inadequate, a volume of blood, representing an undetermined but most probably a very small portion of the contents of the left ventricle, is superadded, and with a force and suddenness equivalent to the contractile energy of the left ventricle. The left auricle, stimulated by distention, reacts with increased energy, and propels the large volume of its contained blood with corresponding force upon the now dilating ventricle. The ventricle, being itself thus surcharged, contracts with vigour, and returns a portion of its contents upon the auricle. The two chambers thus alternately dilate and distend one another, and so become reciprocal stimulants to contraction.

The process thus continued must lead to dilatation and hypertrophy of both cavities, and within a period corresponding to (*a*) the degree of valvular inadequacy, and (*b*) the activity of nutrition. The right ventricle now begins to experience, through the pulmonary vessels, the effects of retrograde pressure from the left side; it undergoes dilatation, and, to a certain extent, hypertrophy, in a corresponding ratio. Flint remarks, that the coexistence of pulmonary emphysema will cause still greater enlargement of the right chambers than mitral inadequacy alone is capable of effecting.\*

Hypertrophy is limited only by nutrition, which, to be adequate, must increase in a ratio proportionate to the growth of the walls. Ultimately, from advancing age, from an inadequate dietary, from excessive stimulation of the heart by labour, alcohol, or emotional excitement, or from defective supply of arterial blood to the walls of the heart, the balance of nutrition

\* *Diseases of the Heart*, second edition, p. 142.



is deranged, and degeneration of tissue quickly follows. Dilatation now proceeds with increased rapidity; blood-stasis and engorgement, first of the pulmonary, and then of the general venous system, ensue; and the last stage, that of dropsy, is close at hand.

Niemeyer held that hypertrophy is consecutive to dilatation of the left ventricle, and that both these changes are of later occurrence than similar changes in the left auricle, because dependent upon them.\* It is manifest, however, that there is, virtually, no pathological sequence whatever, but rather action and reaction, as between the two chambers. That the dropsical accumulations are precipitated by spanæmia from obstruction of the thoracic duct at the acme of venous engorgement, as urged by that author, I can readily admit. It is through this channel that the fat, the fibrin, the nascent corpuscles, and in part the albumen of the blood are introduced into the circulation.

As a consequence of dilatation of the left auricle, the pulmonary veins are enlarged, and their apertures of entrance into the auricle, much dilated; by retrograde pressure, the branches and trunk of the pulmonary artery undergo a similar change. Dr. Milner Fothergill, adopting the opinion of Virchow, believes that atheroma of the pulmonary artery and its branches, is amongst the usual remote consequences of mitral regurgitation, and is due, in his opinion, to interference with the circulation in the bronchial and pulmonary vessels from excessive vascular tension.† Dr. Gerald Yeo has exhibited before the Pathological Society of Dublin an example of extensive atheroma of the pulmonary artery; it was taken from the body of a woman who had died from the consequences of extreme mitral narrowing: "The pulmonary artery, from the semilunar valves down to the very finest subdivision, was irregularly dilated and inelastic; the inner coat throughout being studded with hard, prominent, yellow patches, some of which were rough on the surface, and looked like ordinary atheromatous ulcers."‡

\* *Text-Book of Practical Medicine*, by Humphreys and Hackley, 1869, vol. i., p. 355.

† *The Heart and its Diseases*, 1872, p. 129.

‡ *Proceedings of the Pathological Society of Dublin*, new series, vol. v., part ii. p. 125.

Tension of the pulmonary vessels, though primarily dependent upon reflux from the left side of the heart, is aggravated by the *vis a tergo* from the right ventricle. The irritation to which this latter chamber is subjected from continued engorgement and tension, soon leads to dilatation of its cavity, and thickening, usually but slight, of its walls. On the other hand, these changes in the right ventricle lead by reaction to increased vascular tension in the lungs. Thus, as between the left auricle and ventricle, action and reaction, followed by the usual consequences of mutual stimulation, are exhibited in the relationship of the pulmonary vessels to the right ventricle, in cases of mitral inadequacy. Distention of the right ventricle implies a similar state of the right auricle, parietal congestion of the heart, and engorgement of the trunks and tributaries of the cavæ. The ulterior consequences of these changes are soon exhibited in general venous congestion, enlargement of the liver, stasis of the portal system attended with anorexia, nausea, and foul tongue, and frequently with enlargement of the hæmorrhoidal veins; congestion of the kidneys, with a diminished secretion of concentrated and high-coloured urine loaded with lithates; and finally, anasarca and effusion into the great serous cavities. At a comparatively early stage the tricuspid valves become inadequate by dilatation of the right auriculo-ventricular orifice, and pulsation of the jugular veins is exhibited. This is of two kinds, namely, a strong pulsation synchronous with ventricular systole; and a series of feeble undulations, terminating in a wave of greater magnitude, corresponding to the contractions of the right auricle.\*

Niemeyer, endorsing the statement of Bamberger, maintains that even before tricuspid inadequacy and distention of the jugular veins has taken place, rhythmical undulations of these vessels may be often witnessed as a consequence of transmitted vibration from the tricuspid valve.†

The pulse of mitral regurgitation is in no respect characteristic. Hope maintained that when mitral inadequacy of even slight degree exists, the pulse is always somewhat weak and intermit-

\* See pp. 500 and 571.

† *Opus citat.*, p. 355.

tent.\* Herein he was undoubtedly in error. The pulse is always feeble in proportion to the degree of mitral reflux, but irregularity and intermittence are late characteristics of it, and may be regarded as evidence of failure of the heart. Walshe agrees with Marey in the opinion that coincident mitral stenosis may render the pulse of mitral regurgitation regular, and he further suggests that aortic obstruction may have the same effect. But the pulse, even in the absence of these complications, is usually regular in the early stages of the disease; and in the later stages, it is quite as frequently irregular where they coexist. In fine, I look upon irregularity and intermittence of the pulse, when present in the disease under consideration, as symptoms, not of the valvular inadequacy, but of one of its latest and most formidable sequelæ, debility and failure of the left ventricle.

There are no symptoms suggestive of mitral insufficiency in its early stages, with the exception of palpitation and breathlessness on exertion, and these may be due to so many other causes, pulmonary, hæmic, and nervous, that they must be regarded as possessing very little if any positive value. In the absence of any of these causes, and associated presumptively with a history of antecedent rheumatic affection of the heart, their value will be somewhat enhanced, as pointing to mitral inadequacy.

Congestion of the lungs is amongst the earliest of the advanced symptoms of this affection; it is indicated by dyspnoea and short teasing cough, accompanied by thin serous expectoration; occasionally, there is a streak of blood in the sputum, and, still more rarely, it consists entirely of blood. Hæmoptysis is, according to my experience, by far more common in connexion with mitral narrowing than with simple mitral inadequacy.

All the symptoms, then, which are usually associated with the ulterior stages of mitral regurgitation are no less suggestive of mitral obstruction, and a few of them point even more directly to the latter affection. Hence, the differential diagnosis must rest upon physical signs exclusively. These are, however, sufficiently distinctive, and may be classified under the two heads of signs belonging to the early, and signs appertaining to the later

\* *Diseases of the Heart*, p. 376.

stages of the disease. Murmur and thrill constitute the former group; and these, supplemented by the signs of hypertrophy, dilatation, engorgement, and failure of the heart, make up the latter signs.

Murmur is the most distinctive, and, taken alone, the only pathognomonic sign of mitral regurgitation. It is usually soft and blowing in quality throughout; but occasionally, even in the nascent stage of its development, it is associated with a musical note which is more or less faint, and always, when not substitutive, is terminal in relation to the murmur. At first, and for a more or less protracted period, according to the rate of progress of ulterior changes in the valves and in the heart, the murmur accompanies the first sound, and is, in many cases, at this period postsystolic rather than systolic in rhythm. At a later date, and coincidently with the evidence of utter disorganization of the valves and failure of the left ventricle, it replaces the first sound and becomes substitutive.

In regard to diffusion and transmission, mitral systolic murmur in the adult is strictly limited to the mitral area previous to the development of consecutive hypertrophy, when extrinsic causes of diffusion, such as a solidified lung, do not exist; but in the child, owing to the high conducting properties of the thoracic parietes, it is diffused far beyond these limits, even at the earliest period. In those rare cases in which the valvular lesion is consecutive to hypertrophy from chronic renal disease, atheroma of the aorta, or functional excitement, and where it follows disorganization of the aortic valves, the murmur is quite as extensively diffused from the beginning as it is in the advanced stages of primary mitral lesion.

It has been held by eminent authority that the murmur of mitral regurgitation "of structural mechanism" is almost necessarily audible at the angle of the left scapula and in the left interscapular space, and necessarily so in cases of "well-pro-nounced organic regurgitation."\* I can subscribe the former proposition only in the limited sense above indicated; but the latter I accept in its entirety. Dr. Andrew says: "If the murmur be not audible in this region (at or near the inferior angle

\* Walshe, *Diseases of the Heart and Great Vessels*, fourth edition, 1873, p. 94.



of the left scapula), I believe that it rarely indicates regurgitation."\* Murmur was heard in this situation in 66 of the 100 cases of mitral regurgitation reported by him; but in 64 of these he admits that hypertrophy likewise existed.

Of the 25 cases given in Table XIV., p. 987, most of which were noted many years ago, and before I had been impressed with the value of left scapular murmur as a sign of mitral regurgitation, no record of this sign was made, I regret to say, in 12 instances. It was present in 9 of the cases, all of which likewise exhibited indubitable evidence of hypertrophy, whilst it was absent in 4; and of these, all, with one exception (No. 22), were recent cases.† In the exceptional instance just referred to, there was likewise mitral obstruction, and *hypertrophy did not exist*.‡

I therefore hold, that whilst the existence of a systolic murmur at the angle of the left scapula, coinciding with one of the same rhythm at the apex of the heart, must be regarded as affording confirmatory evidence of mitral regurgitation with hypertrophy of the left ventricle, the absence of it is quite consistent with mitral inadequacy of organic mechanism before hypertrophy has supervened.

Accentuation of the second sound in the pulmonary artery has been held to be an all but essential sign of mitral regurgitation. Skoda so regarded it, and in the absence of this sign, he would have sought an explanation of systolic mitral murmur in assumed roughness of the mitral valve on its ventricular surface, or of the endocardium in the vicinity of the aortic orifice.§

He adds, "An increase in the second sound of the pulmonary artery almost invariably accompanies defect of the mitral valve. I believe that it can only fail to do so when the coats of the pulmonary artery have lost their elasticity, and do not contract rapidly after distention."||

Doctor Walshe does not regard this sign as necessarily present in mitral regurgitation. He declares that he has often known it

\* *St. Bartholomew's Hospital Reports*, vol. i., p. 15.

† Nos. 18, 19, and 21.

‡ See Case 102; also p. 232.

§ *On Auscultation*, by Markham, p. 229.

|| *Opus citat.*, p. 230.

to be wanting where mitral inadequacy existed, and tricuspid regurgitation could not have been adduced to explain its absence on the assumption of diminished pressure.\* The pulmonary second sound was intensified in no less than 70 out of the 100 cases reported by Dr. Andrew.† Nevertheless, as he judiciously remarks, the intensification may be only relative, the second sound in the aorta being diminished in vigour by reflux at the mitral orifice; or it may be apparent, either from masking of the aortic sound, or from favourable conduction of that of the pulmonic artery. Finally, inasmuch as hypertrophy of the right ventricle is admitted by Skoda to be the condition upon which intensified pulmonary second sound essentially depends, and this may be due to causes other than disease at the mitral orifice, *e.g.*, pulmonary emphysema, chronic bronchitis, or pressure upon the trunk of the pulmonary artery, an accentuated pulmonary second sound may likewise depend upon any of these causes. It should be added, that mitral reflux must fail to produce an intensified sound in the pulmonary artery previously to the development of right ventricular hypertrophy; and that it may fail to have that effect even when associated with hypertrophy, owing to impaired elasticity in the coats of the pulmonary artery, as Skoda remarked. On reference to Table XIV., it will be seen that the phenomenon under consideration existed in 7 only of the 25 cases included.

Fremitus or thrill has been regarded by Hope as a frequent attendant of mitral regurgitation.‡ The declaration, however, with which he accompanies this statement, namely, that thrill is less often associated with obstruction than with inadequacy at the mitral orifice, must raise a doubt as to the accuracy of the diagnosis upon which it was based.

Bertin§ declares that tactile fremitus and hoarse murmur (*bruissement*) are "infallible signs" of contraction of the orifices of the heart. This assertion is too general, as both these signs may, although they rarely do, coincide with regurgitation. Tactile fremitus is of frequent occurrence in connexion with mitral stenosis,|| whilst, of the 25 cases included in Table XIV.,

\* *Opus citat.*, p. 94.

‡ *Opus citat.*, p. 387.

† *Loco citat.*, p. 16.

§ *Traité des Maladies du Cœur*, p. 199.

|| See Tables XI. and XII., pp. 964, 967.

it was present in 2 only. It is, in truth, of exceptional occurrence in connexion with simple mitral inadequacy, as might have been presumed from the character of the lesion to which defect of the mitral valve is ordinarily due. When present, it depends upon a rugged and rigid state of the valves, or upon a flake of fibrin entangled in the orifice, and vibrating in the re-fluent stream. In the former of these contingencies the associated murmur would be harsh, and in the latter it would be soft in quality.\*

The evidence upon which the diagnosis of hypertrophy and dilatation of the ventricles should rest has been already pointed out;† the signs of enlargement of the left auricle, however, demand a special notice here. Owing to the deep-seated position of this chamber, the tactile phenomena, indicative of increase in its volume or in the force of its contraction, are of necessity but imperfectly pronounced, and liable to be misinterpreted. A strong pulsation with increased dulness in the second or third intercostal space, immediately inside the left nipple-line, has been regarded as distinctive of dilated hypertrophy of the left auricle; if this pulsation coincide with the presystole, it is held to indicate enlargement of the auricle due to mitral obstruction; if it be systolic in rhythm, it is regarded as diagnostic of a similar condition of the auricle from mitral reflux. I must confess that I am not satisfied as to the alleged value of these signs. The coincident enlargement of the ventricles, or of either of them, must render diagnosis based upon the special localization of the left auricle, very uncertain; and, indeed, the doubtful aid to diagnosis which the phenomena in question are capable of affording in ordinary examples of either lesion can be well dispensed with. In extreme cases they may be so well pronounced as to leave no room to doubt their significance. A case in which they were of this character has been published by Dr. Stokes. A girl, aged eleven years, who had had a well pronounced mitral regurgitant murmur, and was subject to attacks of cardiac asthma, was visited by Dr. Stokes in one of these attacks, which was of unusual severity, and attended with extreme rapidity of pulse and of respiration; the left side of the chest was absolutely dull, ex-

\* *Vide* Nos. 15 and 23 in Table XIV.

† See p. 493, and p. 561.

cept in its posterior and inferior portion, whilst the action of the heart was in the highest degree irregular. The positive and negative evidence led Dr. Stokes to conclude that the dulness of the side was due to temporary but extreme distention and enlargement of the left auricle.\*

Mitral regurgitation, accompanied by murmur, although most frequently due to thickening or shortening of the valves, or vegetations upon their edges,† has been likewise attributed to the several causes mentioned at p. 973. I have not met with examples of mitral reflux murmur, arising from the causes mentioned by Niemeyer, Walshe, and Bristowe.‡ I have already shown (p. 289) that it is scarcely possible to mistake for a murmur of mitral regurgitation, one due to roughening of the ventricular surface of the mitral valve, or of the surface of the ventricle near the orifice of the aorta. The murmur in such a case, although audible at the apex, would be of maximum intensity at the base, and audible by transmission in the ascending aorta and its primary branches.§ A systolic murmur of a musical character, occupying the area and the line of transmission just indicated, has been attributed by Dr. R. St. John Mayne to the vibration of an irregular tendinous chord stretched across the mouth of the aorta, and attached by both its extremities to the walls of the ventricle. In this case, however, the aortic orifice was obstructed by calcareous change of the sigmoid valves.||

Cysts may be formed upon the mitral valve, and give rise to a murmur of inadequacy. An example of this kind, which proved rapidly fatal by rupture of the cyst and septicæmia, has been reported by Dr. Day of Dulwich.¶

Inadequacy of the mitral valve suddenly produced by rupture of one of its cusps, or of the attached tendons or papillary muscles, can scarcely occur where antecedent degenerative changes of the structures involved have not been in operation. The symptoms are those of acute congestion of the lungs by urgent

\* *Diseases of the Heart and Aorta*, p. 204.

† See p. 233.

‡ See p. 974. Various other alleged causes of mitral reflux murmur have been mentioned by authors. See pp. 243, 275, and 280.

§ See Case 31, p. 553.

|| *Proceedings of the Pathological Society of Dublin*, vol. iv., part i., p. 27.

¶ *Biennial Retrospect of Medicine and Surgery*, 1867.



dyspnœa, oppression, and hæmoptysis, accompanied by weakness and irregularity of the pulse, and remotely followed by anasarca. The pulmonary symptoms, as justly remarked by Dr. Todd,\* are more urgent in connexion with rupture of the mitral valve or its sustaining appendages, than with a corresponding accident at the tricuspid orifice. A loud systolic bellows-murmur of mitral origin, and accompanied or not by a musical note, constitutes the principal indication of this accident. Murmur of this rhythm and site, not previously existing, and coinciding in development with the symptoms above mentioned, would warrant a positive diagnosis of rupture of the mitral valve or its appurtenances.

*The treatment* of mitral inadequacy should be directed mainly to the consecutive changes of dilatation of the left ventricle and debility of its parietes. The object of treatment should be to avert the latter contingency, upon which failure of the arterial circulation, pulmonary and general venous congestion, and dropsy, directly depend. Whilst moderate and gentle exercise and a generous but non-stimulating diet should be prescribed with a view to the promotion of nutrition, great muscular exertion, especially such as involves strain and temporary suspension of breathing, and the free use of alcohol, must be forbidden. Indulgence in the use of tobacco, tea, and coffee, as eminently enervating, should be kept within moderate limits. Straining at stool must be avoided; and hence the necessity for occasional aperients, and the use of vegetable food and other alimentary substances which tend to avert constipation. Moderate use of good wines of the tonic class, as claret, etc., may be allowed.

Of medicinal agents, iron, digitalis, quinine, and strychnia are the most valuable; the two former may be given in combination, alternately with the two latter. Thus, ℥v-x of the tincture of the perchloride, or of the acetate, of iron, may be administered thrice daily in an ounce of the infusion of quassia or calumba; or grs. ij of quinine with ℥v of the pharmacopœial solution of strychnia may be given in solution, with a few drops of dilute sulphuric acid, at corresponding periods. For the preceding, a teaspoonful of the syrup of the triple phosphates (iron, quinine, and strychnia) may be substituted. The treatment of pulmonary and general congestion, and dropsy, should be con-

\* *Dublin Journal of Medical Science*, new series, vol. v.

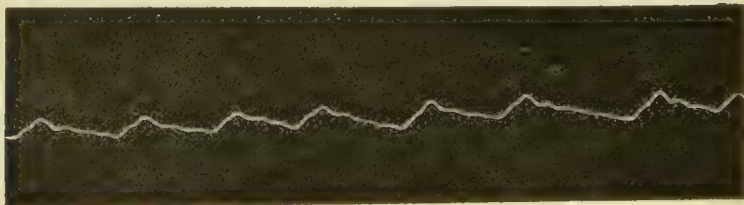
ducted on the ordinary principles. As a diuretic, digitalis, in combination with spirit of nitrous ether, is the best; but as preliminary measures, dry cupping and warm poulticing to the loins, and free purgation, should be resorted to, with a view to the disengorgement of the kidneys.

Hæmoptysis is by no means an ordinary symptom of mitral inadequacy prior to the stage of passive congestion of the lungs; and even then, it is rarely excessive. In this respect, mitral obstruction presents a striking contrast. A streak of blood in the sputa is not, however, an unusual accompaniment of the intercurrent bronchitis of mitral inadequacy.

Of the 25 cases summarized in Table XIV., p. 988, expectoration of blood, even in the smallest quantity, occurred in 7 only. When not excessive, hæmoptysis requires no special treatment; attention should be given mainly to the condition of the lungs upon which it depends. When a styptic is demanded, the liquid extract of ergot, in ℥xv doses, or ergotin used hypodermically, is the most efficacious.

The annexed tracings (Figs. LXV. and LXVI.) exhibit the graphic character of the pulse of mitral regurgitation. Fig. LXV. shows the state of imperfect arterial repletion and defective tension, with regularity of rhythm, which may be witnessed in the early stages of the disease, whilst Fig. LXVI. represents the hypertrophy, with incipient failure of the left ventricle from tissue-degeneration, which characterizes its more advanced stage. (see p. 978.)

FIG. LXV.



Mitral Regurgitation (early stage). Mary M.

FIG. LXVI.



Mitral Regurgitation (late stage). No identification.

No.	Name.	Age.	Occupation.	Hemiphsis.	Dyspnea.	Edema.	Rheumatism.	Irregular or Falling Pulse.	Emptus.	Inten. 2nd Sound in Pulmonary Artery.	Reduplicated 2nd Sound.	Pitch, Quality, and Rhythm of Murmur.	Murmur Audible in Left Back.	Result.
1	Pat. M. ..	24	Turner in wood ..	1	1	1	1	1	..	..	..	Rough and musical, substitutive systolic	*	Discharged.
2	Thomas B. ..	14	Sailor-boy ..	1	..	..	1	..	..	1	..	Soft and accompanying, postsys- tolic	*	Do.
3	Eliza O'B. ..	54	Dressmaker ..	..	..	..	1	1	..	1	..	Systolic, substitutive, and mus- sical	*	Do.
4	Eliza McG. ..	54	*	..	..	..	1	..	..	1	..	Soft, systolic and accompanying	*	Do.
5	Jane E. ..	15	None ..	..	..	..	1	..	..	..	..	Systolic, rough, and accom- panying	*	Do.
6	Jane R. ..	22	Domestic servant ..	1	..	..	1	..	..	..	..	Soft, systolic, and accompanying	*	Do.
7	John McG. ..	22	Merchant's clerk ..	..	..	..	1	..	..	..	..	Soft, systolic, and accompanying	*	Do.
8	Maryanne C. ..	17	None ..	..	1	1	..	..	..	..	..	Soft, systolic, and accompanying	*	Do.
9	Luke McC. ..	63	Butler ..	..	1	1	1	1	..	..	..	Rough and accompanying, post- systolic	1	Death (see Case 121).
10	Susanna F. ..	36	None ..	..	1	1	1	..	..	..	..	Systolic, soft substitutive	1	Discharged.
11	William G. ..	15	Shoemaker ..	..	..	..	1	..	..	1	..	Substitutive systolic <i>b. de scie</i>	1	Death (no <i>p. m.</i> ).
12	John M. ..	21	Van-driver ..	..	..	..	1	1	..	1	..	Soft, systolic, and substitutive	1	Discharged.
13	Lawrence O'B. ..	24	Paper manufacturer ..	..	1	1	1	1	..	..	..	Soft, substitutive systolic	*	Do. (pericarditis).
14	Christopher M. ..	54	Labourer ..	..	1	1	..	..	..	..	..	Soft, systolic, and accompanying, with musical note	1	Do.
15	Catherine O'N. ..	13	None ..	..	..	1	Scar- latina	..	1	..	..	Rough, systolic accompanying	1	Do.
16	George R. ..	12	None ..	..	..	1	1	..	..	..	..	Soft, substitutive systolic	*	Do.
17	Maryanne C. ..	19	Domestic servant ..	1	1	1	..	..	..	..	..	Soft, systolic, and accompanying	*	Death, hypertrophy, and renal disease. (see Case 27).
18	Eliza D. ..	15	None ..	..	..	..	1	..	..	..	..	Musical systolic	..	Discharged. Disease recent.
19	Mary M. ..	60	Domestic servant ..	..	..	..	1	..	..	..	..	Soft, substitutive systolic	..	Discharged.
20	Margaret C. ..	46	Charwoman ..	1	1	1	..	..	..	..	..	Soft, substitutive systolic	*	Do.
21	Anne G. ..	60	Children's maid ..	..	..	..	..	..	..	..	..	Harsh, substitutive systolic	..	Do.
22	Anne K. ..	30	None ..	1	1	1	1	1	..	..	..	Soft, systolic, and accompanying	..	Do.
23	Thomas O'N. ..	15	None ..	..	..	..	1	1	1	1	..	Soft, substitutive systolic	1	Do.
24	Catherine M. ..	42	None ..	..	..	1	1	1	..	..	..	Soft, substitutive systolic	1	Do.
25	Martin M. ..	42	Porter ..	1	1	1	1	1	..	1	..	Soft, substitutive systolic, end- ing in a musical note	1	Do.
25	11 males. 14 females.			7	11	14	16	7	2	7	0		9	Died 3 Discharged 22.

## SUMMARY OF TABLE XIV. (25 CASES.)

Average age of patients, 31 years and 4 months.

						Per cent.
Hæmoptysis	...	...	...	...	...	7 = 28
Dyspnœa	...	..	...	...	...	11 = 44
Œdema	...	...	...	...	...	14 = 56
Antecedent rheumatism			...	...	...	16 = 64
Irregularity or failure of pulse	...		...	...	...	7 = 28
Fremitus	...	..	..	...	..	2 = 8
Accentuation of second sound in pulmonary artery	...					7 = 28
Reduplication of second sound	...		...	...	...	0 = 00
Rhythm of murmur ; systolic	...		...	...	...	23 = 92
„ postsystolic			...	...	...	2 = 8
Quality of murmur ; soft	...		...	...	...	17 = 68
„ „ „ with appended musical note						2 = 8
„ „ rough	...		...	...	...	5 = 20
„ „ „ with appended musical note						1 = 4
„ „ musical	...		...	...	...	3 = 12
Relationship of murmur to first sound ; accompanying						11 = 44
„ „ „ substitutive						14 = 56
Murmur audible in left axilla and back	...		...	...	...	9 = 36
Not stated	...	...	..	...	...	12 = 48

Numerous examples of mitral inadequacy, complicating other forms of cardiac disease, have been incidentally given in preceding chapters.\* The following cases of simple mitral regurgitation are added for the purpose of exhibiting, in an *isolated* form, the symptoms and signs which specially distinguish that affection.

\* The following cases may be referred to ; viz., Case 1, p. 426 ; Case 6, p. 436 ; Case 24, p. 535 ; Case 45, p. 676 ; Case 51, p. 691 ; Case 74, p. 708 ; Case 75, p. 709 ; and Case 76, p. 711.



CASE CXVIII.—*Acute Rheumatism with Hæmoptysis; Precordial Pain; Palpitation and Rough Presystolic Murmur at Apex; Accentuated Second Sound. Diagnosis: Acute Endocarditis with Mitral Regurgitation.*

Thomas B., a sailor-boy, aged fourteen years, was admitted May 23rd, 1865. A month previously he caught cold, which was followed by articular pains, also by pain in the left side and palpitation on exertion. On the day preceding that of admittance, he had spat a good deal of florid blood. When admitted, his condition was as follows: Pulse 96, weak, but regular; heart acted tumultuously; a rough murmur, confined to the area of the apex, succeeded the first sound. The second sound was normal. To have grs. ij of mercury with chalk, thrice daily.

28th. An apex-murmur, of the character and rhythm just described, occupied the entire short pause.

June 1st. Pulse steady, and 102 in erect posture. A sharp grating murmur, strictly confined to the area of the apex, succeeded the first, and ran quite up to the second sound, which was accentuated.

5th. Gums slightly affected by the mercury, which was stopped. An astringent gargle was directed, also grs. v of iodide of potassium, thrice daily.

The boy was discharged towards the end of June, free from urgent symptoms, the apex-murmur, however, retaining its previous characters.

CASE CXIX.—*History of Rheumatism; Palpitation; Great Weakness; Irregularity of Pulse; Displacement of the Apex of the Heart Outwards; Systolic Apex-murmur, which was occasionally attended with a Musical Note; Second Sound Accentuated in the Pulmonary Artery; Repeated Admittances, followed by Temporary Improvement. Diagnosis: Mitral Inadequacy; Hypertrophy, with Dilatation and Tissue-degeneration of Left Ventricle.*

Eliza O'B., a dress-maker, aged fifty-four years, admitted May 15th, 1865. Has suffered occasionally from rheumatic pain in the hips, and about four years ago began to suffer from palpita-

tion and weakness; has never spat blood; is remarkably weak and desponding. Pulse 84, weak, and very irregular. The apex of the heart pulsates an inch and a-half to the outside of, and below the nipple-line; a loud bellows-murmur is heard to accompany the first sound at the apex, and occasionally the latter portion of this murmur is attended with a musical note. The second sound is accentuated at the junction of the third costal cartilage with the sternum on the left side. A bitter tonic with iron was prescribed, with generous diet and stimulants.

She was discharged on the 10th of June, her general condition being then much improved, but the cardiac signs unaltered.

30th. Visited hospital as an extern; was much agitated after a "fright."

She was re-admitted in the early part of May, 1866, being then in a state of great debility; there was slight bronchitis. Action of heart irregular and intermittent; systolic murmur at left apex.

Discharged, at her own request, May 16th. She then felt much better, but the action of the heart was still irregular, and a murmur, as previously described, was audible at the apex.

29th. Admitted again in a state of extreme debility. Pulse 90, weak, and remarkably irregular. The action of the heart was likewise irregular, and occasionally its contractions were not represented in the radial artery.

She was discharged on the 10th of June. There was then no dyspnoea or oedema, but the physical signs were as last described.

This patient was admitted for the last time on the 22nd of June, in a state of extreme weakness, and discharged on the 28th, in a somewhat improved condition.

CASE CXX.—*Displacement of Apex of Heart Outwards; Precordial Pain and Distress; Extension of Precordial Dulness; Rough Systolic Murmur, with Suppression of Second Sound at Apex; Accentuated Second Sound at Base. Diagnosis: Hypertrophy of Left Ventricle; Roughening and Inadequacy of Mitral Valve.*

Jane E., aged fifteen years, admitted June 23rd, 1865. Never

had rheumatism, and never spat blood. Eight months previously she began to suffer from palpitation, and six months later from cough. Pulse 90, regular. Precordial dulness four and a-half inches vertically, by four inches transversely. Apex-beat removed somewhat to left. Urgent precordial pain; cardiac impulse strong; no fremitus; a loud jarring murmur is heard with the first sound at the apex, where no second sound was audible. At the base, both sounds were heard without murmur; the first ill pronounced, the second accentuated. One leech to be applied over heart. To have ℥v of chloric ether in half an ounce of camphor-water every fourth hour.

30th. Is free from cardiac pain and distress, and can lie on left side; murmur less rough.

This patient passed under the care of my colleague, Dr. Hughes, on the 1st of July.

She was re-admitted on the 1st of November following. The feet were then swollen; she had cough, and was in a state of urgent distress from cardiac asthma. Pulse 132, regular, but small. Physical signs as before. Leeches were applied over the heart; and iron, with tincture of digitalis, was given.

CASE CXXI.—*Rheumatic Gout; Tortuosity and Visible Pulsation of the Radial Arteries; Intermittence of the Heart; Rough Postsystolic Murmur at Apex, and likewise audible in Left Back; Second Sound Suppressed at Apex, but Accentuated in the Pulmonary Artery; Re-admittance Seven Years later; Edema of Feet and Legs; Great Debility; Death. Autopsy: Effusion into all the Serous Cavities; Congestion of Lungs and Condensation of the Right with Investment by False Membrane; Enlargement of the Heart; Hypertrophy of both Ventricles; Thrombosis of the Right Chambers and of the Pulmonary Artery; Inadequacy by Thickening of the Mitral Valve; Atheroma of the Aorta.*

Luke McC., aged sixty-three years, an officer's servant, and had served in many climates, was received into hospital February 17th, 1866. Had been subject to rheumatic gout for the last two years.

When admitted, he was suffering from slight pain and swelling in ball of left great toe, and joints of right hand. The tongue was clean; the pulse 72, and slightly visible in the radial arteries, which were rigid and tortuous. Precordial dulness was not extended, and there was no fremitus. The heart acted regularly, and in the normal position. A loud systolic bellows-murmur was heard at the apex; it was extensively transmitted over the left side, was feebly audible in the back, and less distinctly so in the course of the aorta. No second sound was anywhere heard. To have ℞ of tincture of colchicum in an ounce of Murray's fluid magnesia, every third hour.

23rd. The heart occasionally intermitted; urine clear, acid, 1.020 in sp. gr., and free from albumen; skin moist; had then no pain in feet. The murmur at the apex was remarkably rough, and was *preceded* by the impulse of the heart. No second sound was audible at the apex, but it existed at the base, and was accentuated in the pulmonary artery.

26th. The murmur at the apex was distinctly postsystolic, immediately succeeding the impulse and the valve-click of the first sound. There was an occasional intermission, and also from time to time, an irregular and abortive impulse of the heart. Pulse 78. The man was then quite free from pain, and was discharged.

After an absence of more than seven years he was re-admitted in the early part of October, 1873, in a state of great debility. The feet and legs were then swollen. The breathing was occasionally paroxysmal. Urine normal. The heart acted irregularly; the impulse was weak, and the apex-pulsation was felt in the nipple-line; the sounds were faint, but *free from murmur*.

He died on the 14th of October. For several days preceding death, the radial pulse had been scarcely perceptible.

All the serous cavities were found full of serum. Both lungs were congested, and the right was condensed, and covered with old false membrane. The heart weighed twelve ounces, and the right chambers contained some blood-clot. From the right auricle a long cylinder of white fibrin passed into the right ventricle, and thence into the pulmonary artery.

Both ventricles were in a state of hypertrophy, the right being



half an inch, and the left three-fourths of an inch thick. The valves were healthy, with the exception of the anterior and right segment of the mitral, which was thick on the edge. The aorta exhibited patches of atheroma at the root, in the sinuses, and in the transverse portion of the arch.

CASE CXXII.—*Rheumatism ; Chronic Cough, with Hæmoptysis ; Dyspnœa ; Partial Syncope ; Œdema ; Irregularity of Pulse and of Respiration ; Visible Pulsation of Superficial Arteries, and Venous Pulsation in Neck ; Hepatic and Pulmonary Congestion, and Right Pleural Effusion ; Extension of Precordial Dulness, and, Nineteen Months later, Abolition of it from Pulmonary Emphysema ; a Substitutive Systolic Bellows-murmur Terminated by a Musical Note at Apex, likewise Audible in Left Back, all over Left Side, and in the Course of the Ascending Aorta ; Second Sound Accentuated in the Pulmonary Artery ; Three several Admissions, at Intervals of Six and Twelve Months, followed by Temporary Improvement. Diagnosis : Mitral Inadequacy ; Hypertrophy, with Dilatation of both Ventricles ; Fatty Degeneration of Substance of Heart ; Probable Atheroma of the Aorta.*

Martin M., aged forty-two years, a porter in the city fish market, was admitted into hospital on the 26th of April, 1872. Had rheumatic inflammation of the joints two years ago ; for the last three months has been subject to cough, dyspnœa on exertion, and occasionally to faintness, which has obliged him to sit down. The feet began to swell a week before admittance, up to which date he had continued at his work.

On the 27th he was very weak ; respiration was quick, shallow, and irregular ; the pulse was arhythmically irregular, and very feeble ; the pulsation of the radial and carotid arteries was slightly visible ; and there was venous pulsation synchronous with ventricular systole in the supraclavicular and suprasternal fossæ. The feet, legs, and genitals were swollen. The liver was enlarged and tender, and both lungs were congested posteriorly. Precordial dulness extended transversely from the nipple-line to the right margin of the sternum ; the cardiac impulse was dif-

fused and very feeble. At the point of apex-pulsation, which was in the nipple-line, a soft substitutive systolic murmur terminated by a musical note, was heard. At the right base both cardiac sounds were dull, but free from murmur; and at the left base the second sound was intensified. To have, thrice daily, tincture of digitalis, tincture of the perchloride of iron, and spirit of nitrous ether, of each ℥x, in an ounce of infusion of quassia. After a few weeks in hospital the man was free from dyspnoea and faintness; oedema had entirely disappeared, and he returned to his employment.

Re-admitted on the 8th of November, 1872. The lower limbs were swollen; the pulse very irregular; apex-pulsation was felt in the nipple-line. A loud, blowing, systolic murmur was heard at the apex, and at the base, a faint, transmitted murmur of the same rhythm; there was venous pulsation in the neck. After a short course of treatment, similar to that previously mentioned, he was relieved from his urgent symptoms, and was again discharged at his own request, in order to resume his work as a porter.

Admitted, for the third time, on the 2nd of December, 1873. The symptoms then were as follows: Cough and expectoration of mucus, which was occasionally streaked with blood; respiration 36 and shallow; pulse 72, failing, and irregular; partial effusion into right pleura, and oedema of left lung; downward displacement of liver; urine about sixteen ounces in the twenty-four hours. Physical signs: Precordium resonant, and cardiac impulse felt at xyphoid cartilage; a loud, musical, systolic murmur over the left side of the chest generally, in the left back, and likewise in the course of the ascending aorta; it was not transmitted into the arteries of the neck. The point of maximum intensity of this murmur was in the fifth intercostal space, half an inch inside the nipple-line. The carotids pulsated visibly, but there was no venous pulse in the neck.

He was discharged on the 20th of January, 1874, improved as to general symptoms, the physical signs, however, being as last described.

CASE CXXIII.—*Repeated Attacks of Rheumatism ; Strong Pulsation of Heart, and Loud Systolic Murmur at Apex and in Left Back ; Five Years later, Sudden Occurrence of Right Hemiplegia and Amnesic Aphasia ; Pulse Regular, but Dicrotus ; Ecthymatous Eruption on Face and Neck ; Partial Recovery of Motion in Paralyzed Limbs ; Syncope ; Death. No Autopsy. Diagnosis : Mitral Inadequacy of Rheumatic Origin, and subsequent Embolism.*

William G., aged fifteen years, a shoemaker's apprentice, was received into hospital, November 14th, 1861. He had had several attacks of acute rheumatism in childhood ; was pale and haggard, and not grown in proportion to his years ; heart's action strong, and visible pulsation in usual situation of apex. A loud systolic murmur was audible at the apex, and likewise in the left back ; the second sound was normal. He was discharged on the 28th December, being then able to resume his work.

In the course of the succeeding five years, this boy repeatedly presented himself at the hospital as an extern patient, and I had many opportunities of examining him. Within that period his condition underwent little change, except that the point of apex-pulsation of the heart was gradually displaced to the left, and the apex-murmur had become harsh.

Re-admitted on the 31st of October, 1866. Four weeks previous to that date, he suffered much fatigue in attendance on his mother in her last illness, and his feet became swollen. A week subsequently he was found one morning in bed, completely paralyzed on the right side, and incapable of uttering a word. He was then put under treatment by the dispensary medical officer, and took mercury till his gums were affected. His actual condition at the date of admittance was the following: Pulse 98, slightly dicrotus, but regular, and of moderately good volume; *rima palpebrarum* slightly larger on right side than on left; pupils equal; tongue protruded to the right, and with vigour; deglutition unaffected. There was complete loss of motor power in the right arm and leg, without impairment of sensibility. The rectum and bladder were unaffected, and vision was not impaired. He was only able to utter the words "yes," "no," "I do," and a



few others, intelligibly, but with great difficulty, in answer to questions put to him.

When asked his name, he tried hard to remember it, corrugating his forehead and eyebrows in the effort, and seemed angry at his failure. When asked was it "Griffin," he at once exclaimed, but indistinctly, "Griffith," which was his name. In reply to a question as to his age, he said, very indistinctly, "a hundred and eighty." I then asked him was he nineteen, and he at once answered "yes."

The action of the heart was strong, and visible in the sixth and seventh intercostal spaces, a short distance inside the nipple-line; at the apex-point the first sound was replaced by a loud *bruit de scie*; this was likewise audible in the axilla, but not at the base, or in the course of the aorta. The second sound was normal at the right base, but accentuated in the pulmonary artery. To have, thrice daily, grs. iij of iodide of potassium, with ℥x of chloric ether, in a tablespoonful of infusion of cinchona.

On the 3rd of November a few vesicles appeared on the left side of the upper lip; on the following day the eruption had extended somewhat over the left cheek, the left inferior eyelid and *ala nasi*, and had become confluent and opaque.

On the 5th, the corresponding parts of the right side of the face were affected by the eruption, which had now extended much on the left side, and partially involved the neck; it consisted of large round pustules, surrounded by a red areola, and clearly resembling those of small-pox, but not like them depressed in the centre. It presented an ecthymatous character. The conjunctivæ were injected, and the eyes were weeping. The patient's general condition was in no degree affected by it; there were no febrile symptoms, and his appetite was rather improved.

13th. Left angle of mouth much retracted and elevated when the patient attempts to cough. A small blister was applied on the left side of the forehead and temple.

On the 19th he was able to walk across the ward, but with a tottering gait, and his articulation was improved.

On the 3rd of December, after a few days of malaise, a fresh crop of the eruption appeared; it commenced where the blister



had been applied, and soon extended over the entire face and neck; it was confluent in several places, forming patches as large as a shilling, and surrounded by bright-red areolæ. There was, however, no fever. He desired to have wine and chicken, and tried to remember the proper words in which to request them; but failing, he became much excited, placed his hand upon his forehead, and passed into a reverie.

On the 11th, he could raise his right arm nine inches from his side, but there was no improvement in articulation. He was soon afterwards discharged.

He was admitted, for the third time, on the 13th of May, 1867, for a fresh eruption of ecthyma on the face and neck. There was then slight improvement in the power of articulating; he was able to utter two words consecutively. His power of comprehension seemed perfect, and he was manifestly irritated at his failure to recall the proper words to express his wants.

A few days preceding the 3rd of June, when he finally left hospital, he had a fainting fit, in which the heart acted tumultuously. At that date the tongue was still protruded to the right, and he walked with a "drag" of the right leg; there was no further improvement in the motor power of the arm, but in both limbs sensibility was perfect.

He died at his home on the 8th, and I failed to obtain permission to examine the body.

I have reported this case at length, owing to the many interesting features which it presents. The paralysis was manifestly of embolic origin, and the ecthymatous eruption was probably a result of the cerebral lesion.

*Valvular disease on the right side of the heart*, though infrequent relatively to that on the left side,\* is by no means absolutely rare. I quite concur with Dr. Stokes in the opinion that lesion of the valves on the right side of the heart is rarely unaccompanied by a similar affection of the corresponding valves on the left side;† but the existence of valvular disease on the left side, by no means implies a corresponding affection on the

\* See p. 240.

† *Diseases of the Heart and Aorta*, p. 164.

right side of the heart; the latter is related to the former as an exceptional sequence, not as Dr. Latham believed, in the proportion of 1 in 3.\*

Bertin believed that whilst the structural alteration of the valves on the right side is usually fibro-cartilaginous, ossific transformation may be occasionally witnessed; but generally in cases where abnormal communication between the two sides of the heart has existed, the contact of arterial blood having determined a more active inflammatory process.† It is not necessary to remark that ossific change of structure affords no criterion of the activity of antecedent inflammation, nor, indeed, can it be accepted as proof of the occurrence of inflammation in the ordinary sense.

*Disease of the pulmonary valves* is of extreme rarity. Bertin has published an example‡ in which the orifice of the pulmonary artery was nearly occluded by a horizontal septum, formed by the diseased and agglutinated valves, which presented an aperture of only two and a-half lines in diameter. The foramen ovale was patent. The only physical sign observed was precordial fremitus of systolic rhythm. He mentions a case in which obstruction of the pulmonary artery was caused by a rigid tricuspid valve; a systolic murmur, to the left of the sternum, existed in this case.

According to Hope, systolic murmur in the pulmonary artery is more frequently due to extrinsic causes, than to lesion of the valves.§ The accuracy of this statement must be admitted; but that structural lesion of the pulmonic valves, involving both obstruction and inadequacy, may exist, and that it is actually within the range of positive diagnosis, the clinical records published since the date of Hope's work, conclusively show. He has, himself, given the details of a case (p. 599) in which double murmur, accompanied by fremitus, existed in the site of the pulmonary artery; the systolic murmur was remarkably loud, and

\* *London Medical Gazette*, vol. iii., December 27th, 1828.

† *Traité des Maladies du Cœur*, p. 217.

‡ *LVIe Observation*.

§ *Opus citat.*, p. 76.

transmitted in the course of the pulmonary artery, whilst the diastolic murmur was heard in the line of the right ventricle.

Doctor E. L. Ormerod has published three cases in which the physical signs of disease of the pulmonic valves were identified during life, and on *post mortem* examination, the valves were found extensively disorganized.\* These cases are briefly as follows :

1. A woman, aged twenty-one years, was under Mr. Stanley's treatment for syphilis ; a loud systolic murmur was detected, of maximum intensity in a line extending for two to two and a-half inches from the inner portion of the third left intercostal space towards the middle of the left clavicle, but extensively diffused both anteriorly and posteriorly. All the valves were found to be in a healthy state, except those of the pulmonary artery, which were in an advanced state of disease.
2. A man, aged twenty-eight years, under the care of Dr. Bond of Cambridge, had been subject for years to palpitation and dyspnoea on exertion ; a loud and harsh systolic murmur was heard, as mentioned in the preceding case, and shortly before death a diastolic murmur was likewise detected. The pulmonic valves were rolled up, and the orifice barely admitted the passage of a quill. All the other valves were healthy.
3. A man, aged twenty-two years, dropsical, and suffering from dyspnoea, was under Dr. Burrows' care in St. Bartholomew's Hospital. A purring tremor existed over the entire precordium ; the impulse of the heart was strong, and a loud harsh double murmur, most distinct at the junction of the fourth left costal cartilage with the sternum, was likewise detected. A systolic murmur was heard at the ensiform cartilage, and in the line of the right ventricle.

A positive diagnosis of lesion of the pulmonary valves was made in the three foregoing cases.

Doctor Gordon's case,† in which the twofold lesion of the pul-

\* *Edinburgh Medical and Surgical Journal*, vol. lxx., 1846.

† *Stokes on Diseases of the Heart*, Case 19, p. 166.

monic valves existed, and was characterized clinically by a double murmur in the site of the pulmonary artery, audible likewise in the back, but not in the arteries of the neck, and unattended with visible pulsation of the arteries, was one of the earliest and most valuable contributions to the literature of this subject.

Doctor Warburton Begbie has published the details of a typical example of the twofold lesion of the pulmonic valves.\* A man, aged eighteen years, who had been delicate from childhood, and easily put out of breath, but had not had rheumatism or hæmoptysis, was treated for cough, which was not accompanied with expectoration or lividity. Respiration was feeble; precordial dulness, cardiac impulse, and seat of apex-pulsation were normal. A loud, blowing, systolic murmur was audible over the upper portion of the chest generally; it was not transmitted into the carotids, and was loudest at the left third costal cartilage, near the sternum, where a diastolic murmur of much less intensity, and a thrill, likewise existed. The pulse was 72, regular, and not visible. The precordium was prominent, and the muscles of the left arm and chest were less developed than the corresponding muscles of the right side. The man had been under Dr. Begbie's observation during the last three years of his life; he died of fracture of the skull, resulting from a fall whilst in a state of intoxication. The valves of the pulmonary artery were four in number, and incompetent. All the other valves were normal and healthy. This was, most probably, a case of congenital defect of the valves.

A most interesting example of the same kind, though not verified by autopsy, has been recorded by Professor Lebert of Breslau.† A boy of four years, whose breathing had been embarrassed from the first month after birth, had been cyanosed after the fourteenth month, when he began to walk; at the inner portion of the left third intercostal space a loud systolic bellows-murmur was heard; it was likewise faintly audible in the second and fourth interspaces, but in no other situation. The author refers to five examples of congenital stenosis of the infundibulum of the right ventricle, first described by

\* Beale's *Archives of Medicine*, No. 5.

† *Medical Times and Gazette*, January 1st, 1870.



Dittricht, which he had found recorded in medical literature, including one published by himself. In these latter cases he believes the primary disease to have been myocarditis occurring *in utero*; in some cases he believes it to have been of syphilitic origin.

Irrespectively of structural change of the orifice or the valves, other causes of obstruction in the pulmonary artery are occasionally met with. Thus, the pressure of an aneurism; a solid growth within the pericardium, as noticed by Elliotson, or in the anterior mediastinum; or of enlarged bronchial glands, or of a solidified lung, as observed by Latham and Da Costa, may be a cause of obstruction and of systolic murmur in the pulmonary artery.

A case in which an aneurismal pouch, or, at least, one containing blood, situate between the aorta and the pulmonary artery, and causing loud systolic murmur in the latter vessel by pressure, has been recorded by Dr. Flint.\*

Doctor G. W. Balfour concludes from clinical observation, that systolic murmur in the left second intercostal space may depend upon two other causes, irrespectively of disease of the pulmonic valves:† (*a*) the propagation into the left auricular appendix of a mitral reflux murmur of anæmic or organic origin, as first pointed out by Naunyn, or a murmur of mitral narrowing similarly propagated; and (*b*), the pressure and flattening of the pulmonary artery against the anterior thoracic wall, by the systole of the ventricles, the left lung having been permanently retracted by antecedent disease, as alleged by Quincke; in this case the pulmonary artery would be the seat of the murmur.

In reference to the first mentioned cause, I would only remark that a murmur of mitral reflux should be readily distinguishable from all others by its special localization and diffusion; and that the presystolic murmur of mitral stenosis is rarely audible in the second left intercostal space, and when audible there, it may be identified by its preceding the first sound and the impulse of the heart.

As to the second alleged cause of murmur in this situation, if the theory upon which it is based were valid, visible pulsation

\* *Diseases of the Heart*, second edition, p. 243.

† *Medical Times and Gazette*, December 12th, 1874.

in the pulmonic area should be invariably attended by systolic murmur. Yet such is certainly not the case. Indeed, pulsation in the left second interspace, unaccompanied by murmur, is not uncommon in cases of mitral narrowing associated with dilatation and distention of the pulmonary artery and right ventricle, and to this latter condition I believe the pulsation is due.

I have met with an example of partial occlusion of the pulmonary artery beyond the valves, by the pressure and ingrowth of a large cancerous tumor originating in the anterior mediastinum, and involving the lungs.\* In this case a loud systolic murmur was audible over the entire precordium, but most intense in the left second interspace near the sternum.

The recorded examples of pulmonic obstructive murmur due to the pressure of a solidified, and preeminently of a tuberculosed, lung, are more numerous. I have repeatedly met with such, but have kept notes of one only. A discharged soldier, suffering from cirrhosis of the liver and ascites, was received into hospital in October, 1874. It was found necessary to perform paracentesis in order to relieve him from embarrassment of respiration, which constituted his most urgent symptom. This operation was done, for the third time, on the 5th of December; and on that day I discovered partial dulness, with moist crepitant râles over the left front, from the nipple to the clavicle; a loud but soft systolic murmur existed in the internal portion of the second intercostal space on the same side. The area of diffusion of this murmur was limited above by the second costal cartilage, below by the lower margin of the third, and on the inside, by the edge of the sternum. Solidification of the left lung, which was manifestly pneumonic, and the development of murmur, were both of recent occurrence in this case, and most probably in the relation of cause and effect; neither could have existed more than a few days, as the chest had been previously examined with negative results in regard both to the lungs and to the heart. The locality and limitation of the murmur left no doubt in my mind that its seat was in the pulmonary artery.

The man died on the 20th of December. The liver was in an advanced stage of cirrhosis, small, dense, and nodulated, the left

\* See Case 66, p. 734, and Fig. XLI.

lobe being dwarfed in an extreme degree. The spleen was enlarged, the intestines united by false membrane in a few places, and the peritoneum dense and opaque. The heart was structurally normal; the walls of the left ventricle were thick, and its cavity was obliterated by so-called concentric hypertrophy. The valves were all healthy and competent; the pulmonary artery and the aorta were normal as to size and structure, and they contained no clot. The superior lobe of the left lung was dark in colour, dense, and comparatively dull on percussion; it was non-crepitant, but floated in water. The condition was that of hepatized lung-structure in process of resolution.\*

A series of cases of this kind, eight in number, has been published by Dr. Da Costa.† The patients, all young, exhibited indubitable signs of tubercular solidification and softening of the upper portion of the left lung. A loud systolic murmur existed in the pulmonary artery; it was usually soft, but occasionally it was high pitched, and even musical after exertion on the part of the patient, loudest during expiration, and suspended by a full inspiration. This murmur was, in seven of the cases, limited to an area not larger than the bell-end of the stethoscope in the left second intercostal space, close to the sternum; it was not transmitted into the aorta or the carotids, and was accompanied by an accentuated second sound in the pulmonary artery. Dr. Da Costa believes the murmur to have been located in the left branch of the pulmonary artery, and to have been caused by the pressure of the solidified lung.

Murmur in the pulmonary artery may result from thrombosis.

Doctor Gee has reported the case of a young man dying of sun-stroke, in whom he detected a systolic murmur in the pulmonary artery during the last moments of life; the pulse was frequent and feeble, and the temperature in the axilla was 109·5 F. He considered that, owing to the high temperature of the body, a clot had most probably been formed, and impacted in the vessel.‡

Finally, murmurs in the pulmonary artery may be of hæmic

\* This case has come under my notice since the paragraphs on the subject at p. 240 were written.

† *American Journal of the Medical Sciences*, new series, vol. xxxvii.

‡ *Gustonian Lectures*, *British Medical Journal*, March 25th, 1871.



origin. When due to this cause, the murmur would necessarily be systolic in rhythm, as in the cases mentioned at p. 240.

Incompetency of the right sigmoid valves would be necessarily attended with diastolic murmur in the pulmonary artery. I have no personal experience of this condition, which constitutes one of the rarest lesions in pathology.

According to Dr. Cheevers, it is seldom witnessed except as the result of injury, or as a congenital defect.\*

Doctor Bellingham maintains, that the valves of the pulmonary artery present a cribriform condition almost as frequently as do those of the aorta, the latter being usually affected at the same time; but that, owing to the shortness of the pulmonary artery, the feeble reaction of its walls, and in some degree likewise to the seat of the perforations in the valves, a murmur of reflux is not developed.† I entirely dissent from both these propositions; out of many hundred hearts, exhibiting various forms of disease, which I have examined in the *post mortem* room, I have not met with one example of a cribriform state of the pulmonic valves, whilst the records of medicine show that incompetence of these valves is actually attended with diastolic murmur.

In two out of the three cases published by Dr. Ormerod, and likewise in Dr. Gordon's and in Dr. Begbie's cases (pp. 999 and 1000), diastolic murmur existed in the pulmonary artery, and the valves were found incompetent.

I have already (p. 240) pointed out the diagnostic characteristics of systolic murmur arising at the orifice of the pulmonary artery. I may briefly state here that it is distinguished by the following peculiarities, positive and negative. It is remarkably loud and superficial, as correctly noticed by Hope, limited to a small area having its centre at the sternal extremity of the left second intercostal space, and including the adjacent portion of the second and third costal cartilages, but occasionally audible in the interscapular space. Most frequently it is soft and blowing in quality. Negatively, it is characterized by being inaudible to the right of the middle line of the sternum, in the aorta, and in the carotid arteries. By these negative signs it may be distinguished from a murmur of the same rhythm produced at

\* *London Medical Gazette.*

† *Diseases of the Heart*, p. 280.



the orifice of the aorta. From the murmur of an aneurism it must be discriminated by reference to the positive signs of that disease, and especially the circumstance that the murmur of an aneurism, *not* connected with the left extremity of the arch of the aorta, or the descending portion of the vessel, would be traceable, to some extent, towards the right side; whilst, if the aneurism arose from the left portion of the aorta, the effect upon respiratory and percussion-phenomena on the left side of the chest would be sufficiently distinctive.

Inadequacy of the valves at the pulmonic orifice is characterized by a diastolic murmur, most frequently soft and blowing, and transmitted downwards in the line of the right ventricle; that is, towards the ensiform cartilage. From aortic diastolic murmur, that of pulmonary inadequacy may be distinguished, not only by its seat of origin being to the left of the sternum, but likewise, as urged by Drs. Gordon and Begbie, by the absence of visible pulsation in the arteries.

Lesions of the pulmonic valves can rarely be the subject of direct *treatment*, because so seldom the product of acute disease. When detected, defect of these valves from acquired disease is usually so well established, that it is, in regard to the lesion itself, beyond the control of medicine; such is likewise the case when the defect is congenital. The immediate and remote consequences which arise from it, however, namely, surcharge of the general venous system, and defective supply of arterial blood, are in some degree amenable to treatment. This must be conducted on the general principles already pointed out in the discussion of other valvular lesions, and need not be repeated here.

The pulmonary artery is occasionally the subject of *atheromatous change*, and also of *aneurism*. Of the former condition many examples have been published.

Doctor Gerald Yeo has brought under the notice of the Pathological Society of Dublin, a case in which atheroma of the pulmonary artery and its branches throughout both lungs, coincided with constriction of the mitral opening and hypertrophy of the right ventricle.\*

\* *Proceedings of the Pathological Society*, vol. v., part ii., p. 123.

Doctor Rasmussen has published a list of four cases, in which aneurism of a branch of the pulmonary artery caused fatal hæmoptysis by bursting into a cavity in the lung.\* It has been stated by eminent authority that the pulmonary artery is liable to spasm under the operation of certain toxæmic agents.

Doctor George Johnson maintains that, in Asiatic cholera, contraction of the branches of this vessel in the lungs, from the irritation of the cholera poison, is of invariable occurrence in the stage of collapse, and that it explains at once, by reference to the failure of the arterial circulation, suppression of the pulse, depression of temperature, and arrest of secretion; and, on the other hand, by surcharge of the right chambers of the heart and of the systemic veins, that the blue colour of the surface would be accounted for on the same hypothesis.†

Doctor Lauder Brunton has discovered that the dyspnœa attendant on poisoning by *muscarin*, the active principle of the *agaricus muscarius*, is due to contraction of the pulmonary arteries in the lungs, by which the venous side of the heart is engorged, and the arterial side emptied. As the best treatment of mushroom-poisoning, he suggests, after the stomach has been emptied by means of an emetic, that ℥j of the liquor of atropia (B. P.) should be injected hypodermically. This, as he avers, will promptly relax the arterial spasm in the lungs.‡

*Disease of the tricuspid valves*, although comparatively rare, is of more frequent occurrence than valvular lesions at the pulmonary orifice. The tricuspid valves, though structurally healthy, are, however, in the ultimate stages of nearly all other forms of organic disease of the heart, rendered incompetent by dilatation of the right ventricle. Hence, whilst structural alteration of these valves is exceptional, functional incompetence is of constant occurrence. The signs by which this is indicated have been already discussed (p. 238). Here it is only necessary to remark, that the positive signs of tricuspid inadequacy are usu-

\* *Hospitals-Tidende*, Nos. 9 and 13, translated by the late Dr. W. D. Moore. *Edinburgh Medical Journal*, November and December, 1868.

† Pamphlet, 1866.

‡ *British Medical Journal*, November 14th, 1874.

ally limited to the phenomenon of venous palpitation in the neck, where structural alteration of the valve does not likewise exist.

A remarkable example of general venous pulsation in connexion with tricuspid incompetence, has been recorded by Dr. Benson.\* It is necessary to observe, that throbbing of the carotid arteries, in connexion with inadequacy of the aortic valves, or with extreme debility of the left ventricle, if associated, as it frequently is, with congestion of the right chambers of the heart, may be readily mistaken for intrinsic pulsation of the jugular veins. The distinction may, however, be easily made, by pressing the finger lightly upon the distended vein above the clavicle. The effect of this will be to arrest the pulsation, if it be due to reflux from the auricle; whereas, if the pulsation be arterial, this expedient will have no result. It may be further remarked, that the true jugular pulsation is partially arrested by collapse of the veins during *inspiration*, whilst it is exaggerated at the acme of *expiration*. The carotid pulse is in no degree influenced by respiration.

The absence of murmur in cases of simple functional inadequacy may be explained by reference to the debility of the right ventricle, which accompanies dilatation of that chamber. Murmur may, however, be developed in such cases by the engagement of a vibrating shred of fibrin in the orifice. Inadequacy of the valves may be likewise produced by a band of fibrin entangled in the tendinous chords of one or more of the segments, and attached to the wall of the ventricle; but it is usually unaccompanied by murmur.

The valve-segments are, however, occasionally thickened and tuberculated in a degree sufficient to render them inadequate, by antecedent endocarditis, generally rheumatic, and coinciding with a similar affection on the left side of the heart.

Acute inadequacy may likewise result from rupture of the valve, the papillary muscles, or the tendinous chords. Examples of the two former lesions have been recorded by Dr. Da Costa,† and a typical case of the latter accident has been published by

\* *Dublin Journal of Medical Science*, first series, vol. viii., 1836.

† *Toner Lectures*, No. III., Washington, May, 1874.

the late Dr. Todd.\* The symptoms and signs by which it is characterized are sudden dyspnœa, with constrictive pain in the chest, and tendency to syncope. At a later period, engorgement of the liver and kidneys and general dropsy supervene.

Doctor Todd noted repeated hæmatemesis as a symptom in his case, and this he attributes to acute engorgement of the portal system, through congestion of the right auricle. In all these cases the accident has been the result of a sudden wrench or strain, and the injured structures had been presumably unsound when laceration occurred.

Murmur of systolic rhythm and of low pitch is a necessary sign in such cases, because the ventricle, as yet undilated, possesses sufficient contractile force to produce it. The seat of maximum intensity of a tricuspid regurgitant murmur is at the base of the xyphoid cartilage, or slightly to the left of that point; and the line and extent of transmission is upwards and to the right, for a distance of not more than one to two inches.

The limitation of transmission just adverted to constitutes the principal element of differential diagnosis, as between tricuspid and aortic murmurs of systolic rhythm; a further but less constant element is the quality of the murmur, which, when aortic, is usually harsh, whilst it is soft and faint when of tricuspid origin, except in those cases where a vibrating thrombus may have modified its character.

If, with a murmur of the character and area just described, jugular pulsation should be found to coincide, a positive diagnosis of tricuspid reflux may be confidently made.

The phenomenon of venous pulsation in the neck has been already discussed (p. 571). I need only remark here, that it is an infallible indication of tricuspid inadequacy, and, whether accompanied or not by murmur, if the sources of mistaken identity mentioned at p. 1007 be avoided, it may be accepted as proof of tricuspid regurgitation.

*Valvular obstruction* at the tricuspid orifice is usually associated with a similar lesion of the mitral valve, and may be conveniently discussed in that connexion.

\* *Dublin Journal of Medical Science*, new series, vol. v. See also a case by Mr. Squire, *Transactions of London Pathological Society*, vol. xvi., 1864.



Coincident obstructive disease at both auriculo-ventricular orifices is of rare occurrence, yet a few well authenticated examples of it have been recorded. I have myself met with three such examples. Still more rare has been the identification of the twofold lesion during life. Even this, however, has been accomplished in at least three instances.

Narrowing of the tricuspid orifice has been associated with a similar condition of the mitral opening in every recorded example of the lesion that has come under my notice, with the single exception of one published by Bertin.\* It is further to be noticed that in the great majority of the cases, the mitral lesion has been in excess, and in point of time, manifestly in advance of the tricuspid.

Bertin has recorded an example of the twofold lesion which came under his notice in 1814.† The physical signs are not mentioned, nor was a diagnosis attempted.

I find recorded in the Transactions of the London and Dublin Pathological Societies, only nine examples of this lesion, three in the former, and six in the latter. The cases which have been published in the United Kingdom and America, arranged chronologically, are briefly as follows.

In February, 1848, Doctor Quain exhibited the heart of a man, aged thirty-seven, who had acute rheumatism at the age of fifteen years. The most prominent symptoms were cough and hæmoptysis: death occurred by syncope. The lungs were found tuberculous and excavated. The pericardium was adherent to the heart, and, in the false membrane, and likewise in some degree imbedded in the substance of the heart, a calcareous mass was found. The heart weighed fourteen ounces. The segments of the tricuspid valve were thick and opaque, and united for one-third of their extent, the orifice left admitting only the points of two small fingers. The mitral valve and the corresponding chordæ tendineæ were thickened. The mitral orifice was contracted so as to admit only two fingers, and on the auricular surface of the valve were two osteoid nodules. The aortic

\* *Traité des Maladies du Cœur, LVII<sup>e</sup> Observation.*

† *Opus citat., XLIX<sup>e</sup> Observation.*

valves were thickened, and the edge of one of them was everted. No diagnosis of the cardiac lesions was made.\*

In January, 1850, Doctor Pollock reported the case of a woman, aged thirty-eight, who suffered from great dyspnœa, with lividity and general dropsy. There was a systolic murmur, loudest "towards" the apex, but also audible at the base; and over the sternum, at the level of the third rib, a diastolic murmur was likewise heard. She never had rheumatism. The right auricle was found greatly dilated and thickened. The tricuspid valves were united and thickened, forming a diaphragm provided with a central aperture, which admitted only one finger. The right ventricle was reduced in size. The aortic valves were disorganized and incompetent, and the aorta was atheromatous. The mitral valves were thickened, and the orifice so contracted as to admit only one finger. No special diagnosis was made.†

In January, 1852, Dr. Pye Smith submitted the following case: A woman, aged thirty-seven, who never had rheumatism, suffered from dyspnœa, palpitation, and anasarca. The impulse of the heart was strong. A rough systolic murmur, loudest at the apex, was heard, and next, a harsh diastolic apex-murmur. The heart weighed nine ounces. The right auricle was thickened, and enormously dilated. The right ventricle was dilated, and the tricuspid orifice was much contracted. The mitral orifice was greatly contracted, rough, and calcareous, and the attached tendinous chords were thickened. The aortic valves were thick, rough, and inadequate. The diagnosis of auriculo-ventricular narrowing was not made.‡

In 1862 Professor Gairdner made an all but positive diagnosis of tricuspid narrowing in the person of a man, aged twenty years, who suffered no inconvenience from his disease, except the slight amount arising from pulsation of the jugular veins. A presystolic ("auricular-systolic") murmur was detected, and upon this the diagnosis was based.§ The patient died in 1872, and the heart was exhibited by Dr. Gairdner at one of the sectional

\* *Transactions of the Pathological Society of London*, vol. i.

† *Ibid.*, vol. ii.

‡ *Ibid.*, vol. iii.

§ *Clinical Medicine*, 1862, p. 602.

meetings of the British Medical Association in London in the following year. It confirmed his diagnosis, in so far as that an obstacle to the entrance of blood actually existed at the tricuspid orifice; this, however, arose, not from stenosis or from valve-lesion of the orifice, but from the presence of a globular tumor which was attached by a pedicle within the auricle, and floated down upon the orifice after the manner of a ball-valve. This extremely interesting and unique example of disease at the tricuspid opening does not strictly belong to the category of obstructive lesions of the tricuspid valve, although closely allied with them by symptoms and physical signs; the absence of coincident disease at the mitral orifice constitutes, therefore, no exception to the statement above made, in regard to the all but constant association of mitral with tricuspid obstruction.

In December, 1862, Dr. Stokes exhibited before the Pathological Society of Dublin, the heart of a female, aged twenty. The patient never had rheumatism. There had been great variability in the sounds of the heart, and intermittent cyanosis. She was subject to headache and palpitation. A systolic murmur, loudest at the right side of the heart, was occasionally present; it was, however, frequently absent, and a diastolic murmur was occasionally heard. The erect posture increased in an especial manner the variability of the murmur. A month before the patient's death, the murmur was often observed to be presystolic. The carotid arteries pulsated visibly, especially that of the right side, and most strongly in the recumbent posture, and there was a venous thrill at the root of the neck. A murmur was likewise audible in the carotid arteries, and in the left scapular region. At the junction of the third costal cartilage with the sternum, on the right side, a prolonged systolic murmur existed, which was followed by the sounds of the heart. Death took place suddenly by rupture of an intracranial aneurism; and whilst the patient was *in articulo mortis*, a substitutive diastolic murmur was heard. The aortic valves were utterly disorganized; the mitral valves were also diseased, and the mitral orifice was contracted to the diameter of  $5 \times 5$  lines. The tricuspid valves were partially coherent, reducing the diameter of the tricuspid orifice to  $7 \times 6$  lines. The heart weighed twenty-five ounces.



In the foregoing case, an approximate diagnosis was made; a presystolic murmur, which was localized at the right side of the heart, was identified.\*

In April, 1867, Dr. James Little exhibited before the Pathological Society of Dublin the heart of a female, aged twenty-three, who for thirteen years previously had been out of health, and for the last six months especially had suffered from an affection of the chest. The feet were swollen, and the patient had cough, accompanied with dyspnoea and lividity. Evidence of pulmonary emphysema with bronchitis existed. Owing to the extreme respiratory distress, a satisfactory examination of the chest was not made, and no murmur was detected. The right auricle was found to be dilated and thickened, and the right auriculo-ventricular opening reduced to the diameter of the point of the index finger. The left auriculo-ventricular opening was likewise much contracted, but not to an unequal degree with the right. The aortic valves were slightly incompetent. No diagnosis as to the state of the heart was attempted in this case.†

Doctor Balthazar Foster, in the year 1868, observed and reported a case in which he had made the positive diagnosis of tricuspid narrowing.‡ A murmur of presystolic rhythm was detected at the base of the ensiform cartilage; it was, however, loudest to the right of that point, was propagated faintly to the base, but scarcely, if at all, to the apex of the heart. There was pulsation of the jugular veins, recognised as of presystolic rhythm, and fremitus accompanied the murmur at the ensiform cartilage. On *post mortem* examination, the tricuspid orifice was found to be greatly contracted; the mitral opening was likewise, but in a less degree, narrowed.

Doctor Foster remarks that in cases of tricuspid stenosis general venous congestion is greatly in excess of pulmonic. Such is certainly the case, and it constitutes an accessory symptom of much value.

In July, 1869, an example of the twofold lesion came under my notice. A presystolic and a systolic murmur existed at the

\* *Proceedings of the Pathological Society of Dublin*, new series, vol. ii., part i.

† *Ibid.*, vol. iii., part ii.

‡ *Clinical Medicine*, 1874, p. 324.



apex, and the diagnosis of mitral obstruction and regurgitation was made. No murmur was detected elsewhere, and tricuspid obstruction was not suspected. After death, extreme mitral narrowing was found to exist; the tricuspid orifice was likewise partially contracted.\*

Doctor Cryan exhibited a remarkable example of the double lesion in February, 1870. The subject was a female, aged twenty-five. Dr. Cryan made the diagnosis of mitral obstruction and regurgitation; he also diagnosed tricuspid regurgitation, but did not identify tricuspid obstruction. The following remark, which is of the utmost importance in regard to diagnosis, was made by Dr. Cryan: "We could distinguish two seats or centres of murmur, one at the left apex of the heart, and the other at the junction of the fifth right costal cartilage with the sternum."†

Doctor Flint, whilst declaring that a tricuspid direct murmur is "one of the rarest curiosities of medical experience," states that he has met with one example of it. The case was of a complex character, including obstruction at the mitral, tricuspid, and aortic orifice. Doctor Smyth, of Brooklyn, by whom the patient, a young woman, was ordinarily treated, made a presumptive diagnosis of double auriculo-ventricular narrowing, from having heard a presystolic murmur "not only around the apex, but at, and to the right of, the ensiform cartilage."‡

In January, 1871, I submitted to the Pathological Society of Dublin the details of another case of this kind. The patient was a man, aged twenty-three. There was advanced disorganization of the aortic and the mitral valves, involving both obstruction and regurgitation at the respective orifices, all of which had been diagnosed. But, in addition, there was tricuspid narrowing, which had not been recognized.

In reference to this case, I remarked that, "diagnostically the case is of considerable interest. It is perfectly novel to me, and, with the light it affords, I should now have no difficulty in diagnosing, in a similar case, the existence of constriction of the

\* See Case 110, p. 939.

† *Proceedings of Dublin Pathological Society*, new series, vol. iv., part ii., p. 144.

‡ *A Practical Treatise on the Diseases of the Heart*, second edition, p. 240.

two auriculo-ventricular openings. The point on which the diagnosis turns is this, that whereas the murmur of mitral constriction is always at the apex of the heart, and, in the great majority of cases, strictly limited to the area of the mitral opening, in this case a murmur of the same rhythm was audible to the left of the sternum. Between these two points there was a portion of the chest over which no murmur was distinctly audible.\*"

In April, 1872, Dr. Cryan submitted the particulars of another case in which, from the presence of systolic murmur at the apex, and turgescence, with systolic pulsation of the jugular veins, the diagnosis was made, of "mitral insufficiency, combined, *in all probability*, with contraction of the mitral orifice; tricuspid regurgitation, due probably to dilatation of the right auriculo-ventricular opening; hypertrophy, and dilated heart." Both auriculo-ventricular orifices were found to be much contracted, the left in a greater degree than the right; the heart was globular, and weighed eleven ounces.†

The report of the following case, the seventh recorded in Dublin, was communicated by me to the Medical Society of the King and Queen's College of Physicians, in March, 1874. The morbid specimen was, at the same time, exhibited to the members of the Society. A woman, aged twenty-five, for many years out of health, suffered from cough, hæmoptysis, great dyspnœa, and œdema of the lower limbs. There was congestion of the face and neck, and great turgescence of the jugular veins. A presystolic murmur was detected at the apex of the heart, which pulsated to the left of the nipple-line. A murmur of the same rhythm, but harsher, was heard near the left margin of the sternum, in the fourth intercostal space. At a point intermediate to these two seats of murmur, neither was distinctly audible. The diagnosis of both mitral and tricuspid obstruction was made, and confirmed by the autopsy.‡

In November, 1872, a man, aged twenty-five, the subject of double auriculo-ventricular contraction, was admitted into hos-

\* *Proceedings of the Dublin Pathological Society*, vol. iv., part iii.

† *Ibid.*, vol. v., part i.

‡ See Case 116, p. 956.

pital for me. He came from a distant part of the country to obtain relief from palpitation and weakness. He had never had rheumatism, and his health had been good up to six months previously. Since then, he had repeatedly spat blood.

There was no œdema, cough, or dyspnœa, and no engorgement of the cervical veins. He frequently sighed, and declared he experienced relief from the act. The pulse, in the radial artery, could not be registered, owing to its extreme weakness and irregularity. The pulsations of the heart, which were likewise irregular, numbered 120 in the minute. The apex-pulsation was felt in the nipple-line and fifth interspace. At this point a presystolic fremitus was felt, and a loud and harsh presystolic murmur was heard. This latter was loudest at two points; namely, at the apex, and two inches to the inner side of, and somewhat below the left nipple. The second sound was not double or accentuated.

The man's health improved under treatment with iron, digitalis, and strychnine; but the sighing continued, and the physical signs were as above described at the date of his discharge, a fortnight after admittance. I have not since heard of him. The diagnosis of the double lesion was made from the existence of two distinct seats of murmur, accompanied with fremitus.\*

A fifth example of auriculo-ventricular narrowing, on both sides of the heart, came under my notice in July, 1874. The patient was a woman, aged fifty-six, suffering from dyspnœa and œdema, and subject to hæmoptysis. The face and neck were congested, and more so after exertion; they were usually swollen in the morning. A presystolic murmur existed at the apex, and a murmur of the same rhythm was heard in the fourth interspace, near the left margin of the sternum.† The diagnosis was made, as above mentioned; but the opportunity for testing its accuracy by *post mortem* examination, was happily not afforded, as the poor woman improved somewhat under treatment, and left hospital after a fortnight's residence, to resume her duties as a midwifery nurse.

It would seem that Hope had not met with an example of

\* See Case 52 in Table XI.

† See Case 15 in Table XII.

tricuspid narrowing. M. Aran has declared that he himself had not.\*

In 1868, M. Raynaud questioned the possibility of making the differential diagnosis of tricuspid obstruction. He says: "As to morbid sounds of diastolic and presystolic rhythm, we are not convinced that they can be referred, with certainty, to the right side of the heart, and we even doubt that the right auricle possesses sufficient force to produce a murmur immediately preceding the ventricular systole." He adds: "What well-informed practitioner, then, will be bold enough to say, 'This belongs to the right, and that to the left heart?'"†

Amongst the preceding cases will be found at least three, which supply an affirmative answer to this query. These three cases establish, beyond question, the practicability of distinguishing between mitral and tricuspid presystolic murmur.

In reference to treatment, I would only remark that, in addition to the measures recommended in cases of mitral obstruction, all of which are applicable to the double lesion, the abstraction of a few ounces of blood from the arm would afford at least temporary relief to the state of high venous tension which exists in the latter cases.

*Disease of the coronary arteries*, consisting in atheromatous change or calcification of their coats, has been met with most frequently in connexion with similar changes at the root of the aorta, involving the valves, the walls of the vessel, or both. It has been likewise witnessed very frequently in cases of fatty degeneration of the heart.

Doctor Quain found the coronary arteries more or less altered and obstructed in 13 out of 33 cases of fatty degeneration recorded by him, and in 1 case (26, series I.), the branch of supply from the coronary artery to the *only* portion of the heart which exhibited fatty metamorphosis, was completely calcified.‡ The connexion between disease of the coronary arteries and fatty degeneration of the substance of the heart, may be readily ex-

\* *Archives Générales de Médecine*, tom. xv<sup>e</sup>, 1842.

† *Dictionnaire de Médecine et de Chirurgie*, tom. viii<sup>e</sup>, p. 643.

‡ *Medico-Chirurgical Transactions*, vol. xxxiii.



plained by reference to the impairment of cardiac nutrition to which disease of these vessels must lead, on the assumption, which cannot be disputed, that fatty transformation of muscle is an atrophic change. Similar results may, however, follow from simple obstruction of the coronary arteries at their origin, by calcareous deposition in the sinuses of Valsalva. I think it may be assumed, even in the absence of disease of the coronary arteries, or occlusion of their orifices, that fatty disease of the heart is the direct result of a feeble circulation through these vessels, in cases of primary dilatation and atheroma of the first portion of the aorta. In such cases the coronary circulation, which depends mainly, if not entirely, upon the recoil of the aorta, fails, owing to the rigid and inelastic state of the walls of that vessel.

Thrombosis of the coronary arteries may occur in cases of parietal disease of these vessels, and become the cause of acute fatty transformation of the heart. Calcification of these vessels has been regarded as the essential cause of angina pectoris ; but this doctrine is too exclusive, and is not admitted by modern pathologists.

## CHAPTER VIII.

### FORMATION AND MIGRATION OF BLOOD-CLOTS.

THROMBOSIS of the heart, or the formation during life of clots or so-called polypi within its chambers, consisting of separated fibrin and blood corpuscles in a more or less altered condition, may occur as a consequence of: (*a*) The admixture with the blood of foreign matters in a state of minute division, or of organic fluids in process of septic change;\* (*b*) Hyperinosis, as in acute rheumatism; (*c*) High pyrexia, as in the early stage of scarlatina;† (*d*) Retention of excreta, as in chronic renal disease;‡ (*e*) Pyæmia; (*f*) Leukæmia; and (*g*) Protracted death struggle in cardiac, renal, or pulmonary disease.§

Doctor Bristowe concludes that "sex and age have no obvious influence on the formation of cardiac thrombi; that they occur in all the cavities of the heart, but most frequently in the left ventricle, and least frequently in the left auricle; that they affect, with scarcely an exception, those parts of the cavities which are out of the direct line of the circulation, and are always fixed by being entangled amongst the inequalities of surface; that they are met with, almost exclusively, in those forms of disease in which the process of death is, as a rule, protracted; that their arrangement, form, and microscopic characters seem to show that (excluding one or two unusual cases) they consist merely of blood which has coagulated during life, and has undergone those changes alone which clots elsewhere in the living body are liable to undergo; that they are the result of spontaneous coagulation of blood in the heart's cavities, at a variable period anterior to death, but occurring at a time at which the

\* Mr. Savory, *St. Bartholomew's Hospital Reports*, vol. i.

† Dr. John Harley, *Medico-Chirurgical Transactions*, vol. lv.

‡ Dr. Bristowe, *Transactions of Pathological Society of London*, vol. vii., and Dr. John Ogle, *ibid.*, vol. xiv.

§ Bristowe, *loco citat.*

patient is in a moribund condition, or in a condition of threatening dissolution.\*

These masses consist mainly of fibrin more or less completely separated from the other constituents of the blood, and exhibiting various shapes, according to the size to which they have attained, and the positions which they occupy. Most frequently they are smooth on the surface, and of a light buff tint, but occasionally the colour is nearly white. When small, they may be ribbon-shaped, the band extending from the auricular appendix, where it is firmly attached to the pectinate columns, or to a dense mass of the same structure there impacted, through the auriculo-ventricular opening into the ventricle, and sometimes from the ventricle into the pulmonary artery or the aorta. This band not unfrequently renders the respective valves incompetent by being entangled in their edges, or in the tendinous chords; it occasionally, though rarely, takes origin from the apex of either ventricle, proceeding thence towards the arterial orifice, or into the artery. Thrombi of this figure usually coincide with coloured coagulum, and are blood-stained on the surface; but their structure is fibrinous and uniform throughout. Occasionally the auricle or ventricle on either side of the heart is all but completely filled by a large thrombus, which has assumed the shape of the cavity, forming a perfect but reduced cast of it, and leaving only a narrow interspace on the surface through which an imperfect circulation is still maintained.

I have seen both the right chambers and the tricuspid orifice occupied in this manner, whilst the left cavities contained some detached clots and flakes. Massive thrombi of this kind consist of a shell of decolorized fibrin, from half a line to a line thick, and a soft and grumous parenchyma, composed of the *débris* of fibrin and blood corpuscles, and usually tinted red.

Doctor John Ogle has found in these bodies "amorphous molecular and fatty material, imperfect cellular, nuclear, and fibrillary forms, with compound granular corpuscles."† Within a short time, as a consequence of disintegration and putrefactive change, the contents may assume the appearance and the consistence of pus; and, as Dr. Ogle surmises, they may be diffused

\* *Loco citat.*

† *Loco citat.*

in the circulation by rupture of the cyst, and so give rise to symptoms of pyæmia. That the contents of cystic thrombi within the heart are never truly purulent, may be now regarded as proven.

Smaller bodies, composed of fibrin in process of change, and presenting various figures, are constantly found in the heart, even where no obvious symptoms have been attributable to their presence; such are flakes and bands of fibrin attached to the papillary muscles and tendinous chords, and floating loosely in the cavity; small irregular bodies of a solid but friable structure, impacted in the recesses between the fleshy columns; nodular deposits upon the valves; and pedunculated globular masses, from the size of a pea to that of a walnut, suspended from some portion of the wall, as described by Laennec.\* The thrombus may consist in a thick lining of precipitated fibrin, extending throughout both the right chambers, and forming a hollow cast of them, through which the blood had circulated.†

I believe the right chambers of the heart are by many degrees more frequently the seat of thrombosis than the left chambers; a difference for which the less degree of muscular development, and the greater liability to stasis on the right, as compared with the left side of the heart, will supply an adequate explanation. Of the 44 fatal cases of valvular lesion given in Table X. (p. 815), 24 exhibited thrombosis on the right side of the heart, and 12, thrombosis on both sides. In no instance was the left side alone thrombosed.

I have witnessed cardiac thrombosis more frequently in connexion with dilatation and debility of the right chambers of the heart than with any other condition. Obstruction on the left side of the heart, and pulmonary emphysema with chronic bronchitis, issuing in dilatation and distention of the right chambers, have furnished the greatest number of examples. The two essential conditions for precipitation of fibrin from the circulating blood are herein supplied; namely, stasis, and a rough surface.

Irrespectively of symptoms, most valuable evidence of the

\* "Végétations Globuleuses," *Traité d'Ausculation Médiate*, tom. ii., p. 630.

† See Case 110, p. 939.



*ante mortem* formation of these bodies is occasionally furnished by their configuration and surface marking. An impression of the aortic valves, in a state of partial closure, has been found by Sir Dominic Corrigan upon a thrombus extending from the left ventricle into the aorta.\*

I have frequently observed a similar condition of the clot engaging the aortic orifice, and likewise the mitral and tricuspid openings.

The *symptoms* of cardiac thrombosis are, in many cases, sufficiently pronounced to warrant a positive diagnosis. It must, however, be added, that in the majority of instances the evidence available during life will admit of a presumptive diagnosis only; this observation applies to the cases in which bands or small masses, not implicating the valves or the orifices, have been formed.

Extreme thrombosis of the right chambers may be confidently diagnosed from the following symptoms occurring in a case of chronic pulmonary or cardiac disease: Reduction in volume, and increase in the rate of the pulse; faintness; orthopnoea, characterized by a struggle for breath, without difficulty of breathing (air freely enters the lungs, but it does not satisfy the *besoin de respirer*); tumultuous and irregular action, with occasional intermissions of the heart, feeble impulse according to Richardson,† and partial or complete suppression of the sounds on the right side. The area of precordial dulness is extended to the right; the respiratory sound is full, and even sonorous. The nares are dilated, the chest heaves with inspiration, whilst the patient is agitated, moans, and craves for air;‡ the veins of the neck are turgid, the features anxious and livid, and the surface cold, and often humid with perspiration.§

The symptoms of clot on the left side of the heart are of a less distinctive character. Richardson avers that the sounds are faint or suppressed on the left, though audible on the right side, whilst the impulse is heaving and tumultuous. I am not in a

\* *Proceedings of the Pathological Society of Dublin*, new series, vol. ii., part i.

† *Medical Times and Gazette*, November 21st, 1868.

‡ "Summæ anxietates" of Van Sweiten.

§ See Case 106, p. 927; also Cases 107, p. 932; and 111, p. 942.

position either to confirm or deny the accuracy of this observation. The weakness of the radial pulse, however, contrasts with the strength of the cardiac impulse, whilst the signs of pulmonary congestion are well pronounced, and often associated with hæmoptysis.

On either side of the heart, a small and floating thrombus, engaged in one of the orifices, may give rise to a systolic murmur of a more or less musical character.

The *treatment* of thrombosis of the heart must be preventive rather than curative. Whilst we cannot hope to effect solution of the clot already formed, we may succeed in preventing its further growth by medicine. Alkalies afford the best, although a doubtful prospect of accomplishing the latter object.

Doctor Richardson proposes to give, at short intervals, ℞ doses of the *liquor ammoniæ*, in an ounce of water; whilst Professor Gerhardt prefers bicarbonate of soda, which he administers by inhalation, as a more direct mode of introducing it into the heart. The solution used by Gerhardt contains from a-half to one-and-a-half per cent. of the salt, and the results, as reported by him, are encouraging.\*

*Thrombosis of the arteries and veins* is of rare occurrence in the absence of local inflammation of the walls of the vessels; and in these exceptional instances it is usually secondary to an impacted embolus.

Doctor Liddel, however, thinks that arterial thrombosis may result from an excess of fibrin, or of white corpuscles, in the blood; or from atheromatous or calcareous change in the coats of the vessels.†

I have occasionally seen a deposit of fibrin upon the internal surface of the aorta, where roughened and fissured in the process of atheromatous transformation; but in most instances there has been local dilatation of the vessel in the seat of the deposit.

Thrombosis of the veins usually depends upon phlebitis in the seat of the clot. Uterine phlebitis, consecutive to parturition, is the most frequent cause of it; hence it is usually exhibited in the

\* *Deutsches Archiv. für Klinische Medicin*, vol. v., p. 207, summarized in *The Dublin Quarterly Journal of Medical Science*, May, 1869, p. 421.

† *American Journal of Medical Science*, January, 1873.

femoral and internal saphena veins. This is true, not only of the *post partum* form of venous thrombosis, but likewise of this affection arising from other causes, owing to the tendency to stasis in the veins of the lower limbs, as compared with the other portions of the body. These vessels are unfavourably circumstanced in regard to their circulation, not only by having to sustain the weight of the column of blood in the iliac veins and the inferior cava, and to propel their contents against gravity in the upright posture, but also by the casual obstruction to which they are liable from pressure within the abdomen, from stasis in the lungs, or in the right chambers of the heart.

The cerebral sinuses and veins are, next after the veins of the lower extremities, the most liable to thrombosis. This arises, most probably, from the slowness and equability of the circulation, which results from the absence of direct atmospheric pressure within the cranium.

The gravity of venous thrombosis depends upon the danger of consecutive embolism, the seat of which, when the systemic veins are engaged, is the right side of the heart and the pulmonary artery.

Phlebitic thrombosis may arise, not only from mechanical injury to the veins, and the direct introduction of septic poisons into those vessels, as occurs in the female after parturition, but likewise from the operation of certain morbid agents developed in the blood itself, or from changes of its constitution.

Doctor Tuckwell has recently published three cases of phlebitis, followed by embolism of the pulmonary artery, in gouty subjects, produced, as he believes, by the operation of the morbid element of gout in the blood.\* Sudden death occurred in two of these cases; in one of the latter the internal saphena and the femoral veins of one limb were found to have been the primary seat of inflammation and coagulation; the man, who was gouty, was convalescent when he imprudently got out of bed, and, "in the act of bending his thigh to get into bed, he broke off a large piece of the clot in the femoral vein, which rushed into the left pulmonary artery, and suddenly deprived him of the use of his

\* *St. Bartholomew's Hospital Reports*, vol. x., 1874.



left lung," the right lung having been previously and universally infarcted, as shown by the autopsy. The femoral vein, the right ventricle of the heart, and the left pulmonary artery, were occupied by separate portions of a large cylinder of solid fibrin, the broken ends of which accurately fitted to one another. In the third case, typhoid or pyæmic symptoms were developed, which were supposed to have arisen from blood-poisoning by disintegration of the clot; the patient ultimately recovered.

A case of pulmonary embolism, consecutive to phlebitis of the internal saphena in a pregnant female, has been published by Mr. Brudenel Carter. Improvement did not take place, as usual, after confinement, and the patient soon after died quite suddenly. A clot was found in the pulmonary artery.\*

Doctor Tuckwell† also furnishes, in the same article, the particulars of two cases of chlorotic venous thrombosis, one in the lower extremities, and the other in the cerebral veins and the sinuses of the dura mater. In the latter, which was suddenly fatal, nearly all the sinuses and veins of the right hemisphere of the cerebrum were found full of coagulated blood; extravasation and softening, to a slight extent, had likewise taken place.

A case similar to that last mentioned has been reported by Dr. Andrew.‡ Severe and persistent headache, followed by coma, were the chief symptoms in both these cases.

I have met with examples of venous thrombosis of the lower limbs in the last stages of pulmonary phthisis. The following is an example of this kind. John B., aged twenty-seven, a coal heaver, was received into hospital in May, 1869. He had been intemperate, and five months previously he was first taken ill. When admitted, he was suffering from dyspnoea, with cough and expectoration. Both lungs exhibited signs of extensive tubercular softening. A few days after admittance, he complained of pain in the calf of the right leg, the limb became swollen, and likewise tender in the course of the femoral vein. On the 9th the entire limb was livid, greatly swollen and very tender, the femoral vein in Scarpa's triangle, was rigid

\* *Medical Press and Circular*, April 17th, 1872.

† *Loco citat.*

‡ *Reports of Pathological Society of London*, vol. xvi.



and knotty, whilst the femoral artery pulsated strongly in the same situation; the temperature of the limb was not depressed.

The man died soon afterwards, but without having exhibited symptoms of embolism. The right femoral vein was found full of decolorized and solid fibrin, which was firmly attached to the coats of the vessel to the extent of two inches. No other parts were examined. In all such cases, Dr. Tuckwell very properly insists upon the necessity for rest and immobility in the recumbent posture; sudden flexion of the limb should be especially avoided.

*Embolism*, or the migration of clots within the blood-vessels, is a subject which has been recently studied with great earnestness, and has yielded most remarkable results. Many cases of sudden death, and still more numerous examples of sudden hemiplegia without loss of consciousness, and of instantaneous loss of vision in one eye without previous disease of the organ, which, under the pathology of thirty years ago would have been totally inexplicable, may be now demonstrably traced to this accident.

Professor Trousseau\* has assigned to Legroux the merit of having been the first to enunciate this doctrine. To him is due the honour of its revival only; for, as shown by Dr. Walshe, Van Swieten had anticipated him by more than half a century. After the observations of Legroux (1827), came those of Allibert (1828), Louis and Lediberder (1837), and Baron (1838).† Virchow has investigated the subject with his customary exhaustiveness,‡ but the late Dr. Kirkes was the first to establish conclusively the connexion between valvular disease of the heart and embolism.§ He published, in 1852, three cases of suddenly developed hemiplegia consecutive to cardiac disease. In all three the lesion was valvular; mitral alone in two; mitral aortic and tricuspid in one. The valves, in each of these instances, were studded with fibrinous deposits, in the form of vegetations or flakes. The middle cerebral artery of either side was found plugged with

\* *Clinical Lectures*, Sydenham Society's edition, vol. iii., p. 414.

† Walshe, *Diseases of the Heart*, fourth edition, p. 451.

‡ Froriep's *Neue Notizen*, 1846, see p. 910.

§ *Medico-Chirurgical Transactions*, vol. xxxv., 1852.

fragments of fibrin in all these cases, and the corpus striatum and adjacent portions of the brain were softened. The renal and splenic arteries were likewise plugged. In a fourth case, a sacular aneurism of the left ventricle existed; the sac was filled with laminated fibrin, the splenic artery was plugged, and the spleen and kidneys exhibited patches of capillary infarction.

Doctor Bristowe has given the particulars of seven cases of plugging of the cerebral arteries, in two of which it was due to detachment of fibrin from diseased cardiac valves.\*

Doctor Hughlings Jackson has published several examples of hemiplegia in connexion with valvular disease of the heart, in which he had diagnosed cerebral embolism.†

Of 37 cases under Dr. Hammond's care, in which cerebral embolism had been diagnosed, organic disease of the heart existed in all except one; and in 7 of these cases, in which the body was examined after death, the left middle cerebral artery was found to be the seat of an embolus.‡

From the records of the Pathological Institute of Berlin (1868-'71), it appears that emboli existed in 29 per cent. of all the cases of endocarditis; in two or three instances they originated on the right side of the heart, and produced infarction and abscess in the lungs, chiefly in the inferior lobes; in 26 per cent. of the cases the emboli were derived from the left side of the heart; and of these, the mitral valves alone were found to have been their source in the proportion of 87 per cent., and both the mitral and the aortic valves in the proportion of 49 per cent. The kidneys were the seat of embolism in the proportion of 75 per cent.; the spleen, 51 per cent.; the brain, 20 per cent.; the intestinal tract and liver, 7 per cent.; the skin, 5 per cent.; the spinal cord, 3 per cent.; and the thyroid body and the eye in a somewhat less proportion.§

Doctor Olaf Page, of Valparaiso, has published the case of a young man, the subject of organic disease at the aortic orifice, in whom sudden loss of vision was found, on *post mortem* examina-

\* *Transactions of Pathological Society of London*, vol. x.

† *Clinical Lectures and Reports of the London Hospital*, vol. i., 1864.

‡ *A Treatise on the Diseases of the Nervous System*, 1871.

§ *Edinburgh Medical Journal*, July, 1873.

tion, to have been due to embolism of the retinal artery; the left middle cerebral artery was likewise plugged.\*

An example of embolism of the right axillary artery, consecutive to thrombosis of the left auricle or ventricle in a chlorotic female, free from organic disease of the heart, is given by Dr. Tuckwell;† gangrene of the fingers took place, but the patient recovered.

Amongst the cases of valvular disease recorded in the preceding pages of this work, 136 in number, 13 examples of hemiplegia will be found which were clearly of embolic origin. Of these, 8 were on the right side of the body, and 5 on the left; the mitral valves alone were the seat of disease in 7 instances; both the mitral and the aortic valves, in 4; and the aortic valves alone, in 2 cases. I have met with only one example (No. 13, Table XII.) of sudden loss of vision from thrombosis, with retinal hæmorrhage. It would thus appear that the liability to embolic hemiplegia is much greater in connexion with mitral than with aortic valve disease. I may add, that this liability attains its maximum in cases of mitral stenosis.

Plugging of the peripheral branches of the internal carotid artery, by migratory clots detached from the valves of the heart, is readily explained by reference to the direct route of transit which that vessel supplies for these bodies. Hence, the frequent implication of the brain and the eye. It is, however, remarkable, and at present inexplicable, that the left hemisphere of the cerebrum, and the left eye, should be more frequently the seat of these lodgments than the right; yet so it is. In my 13 cases, the proportion of left cerebral thrombosis to right was as 8 : 5.

The middle cerebral artery, being the largest and the most direct of the terminal branches of the internal carotid, is most frequently the seat of the plug; next, in the order of frequency, are the anterior cerebral, and the ophthalmic.

The mechanical result of plugging in the cerebral or retinal arteries is similar to that exhibited in the lungs from embolism of the branches of the pulmonary artery (p. 910); namely, vascular engorgement and extravasation on the distal side of the clot.‡

\* *American Journal of Medical Science*, January, 1874.

† Case 7, *loco citat.*

‡ See Case 111, p. 942



The gravity of the effects of this local disorganization must be necessarily proportionate to, (a) the delicacy of structure, (b) the importance of function, and (c) the inadequacy of the collateral circulation of the part. In the portion of the brain usually affected by an embolus, namely, that supplied with blood by the middle cerebral artery, and in the retina, these three conditions represent a maximum quantity. Hence, suspension and subsequently loss of the function of the corpus striatum and the adjacent portion of the cerebrum, from blocking of the middle cerebral artery ; hence, likewise, similar results in the eye, from embolism of the central artery of the retina.

Professor Trousseau believed that mere occlusion of the middle cerebral artery will not afford an adequate explanation of the results which are apparently connected with it, and that the pressure of the clot upon the cerebral substance must be regarded as an important factor.\* But the position and the minuteness of the clot render the latter view inadmissible, whilst the absence of a collateral circulation in the portion of brain substance supplied by the middle cerebral artery, as correctly pointed out by the late Dr. Kirkes, renders the changes consecutive to closure of this vessel quite intelligible. Complete restoration of structure and of function may take place by early disintegration of the plug, but experience shows that such a result is exceptional.† I have witnessed partial, but never complete, restoration of motion and of vision in cases of embolic hemiplegia and amaurosis. It is worthy of remark, that the paralysis resulting from cerebral embolism affects motion only ; sensibility is unimpaired, and the paralyzed limbs are, in many cases, even hyperæsthetic.

*Capillary embolism*, or *infarctus*, due to impaction of pulverized fibrin in the extreme vessels of a part, is usually attended with less grave consequences than embolism of the larger vessels, owing to the very limited district of tissue which it affects. It is most probable that the occurrence of transient vertigo and loss of vision, with inability to maintain the erect posture, or even momentary loss of consciousness, so frequently witnessed in connexion with valvular disease on the left side of the heart, is due

\* *Opus citat.*, vol. iii., p. 415.

† See Case 88, p. 868.



to capillary embolism of the brain by the detachment of detritus from the diseased valves.\* Hemiplegia, of greater or less duration, accompanied by impairment or loss of speech and of memory when the lesion is located in the left side of the brain, is occasionally the result of infarction of the cerebral capillaries; Case 100 (p. 887) affords a probable example of this kind.

*Embolism of the pulmonary artery* and its branches has been already discussed in regard to its pathology (p. 910).† The migratory clot is derived, most frequently, from the right side of the heart, and is consecutive to cardiac thrombosis; it is occasionally the result of detachment of a clot from an inflamed vein, as previously shown (p. 1023). The gravity of the symptoms of pulmonary embolism will depend upon: (a) the order of the vessel engaged; (b) the size of the plug; and, when the vessels of one side only are affected, (c) the actual state of the opposite lung at the moment of the accident. When it arises from a thrombus formed within the heart, the consecutive changes in the lungs are usually localized, and of limited extent; and the symptoms are those of pulmonary apoplexy, including hæmoptysis. Even in such cases, however, sudden death may occur from infarction of the trunk of the pulmonary artery and its branches in both lungs. Cases 124 and 125 afford examples of this kind. The previous disease, in such cases, is usually constitutional, not simply valvular; most frequently it is leucocythæmia.

When the trunk of the pulmonary artery is plugged, the impacted body is usually solid, and has been moulded in one of the larger veins, whence it has been conveyed by the blood-stream to the right side of the heart, and into the pulmonary artery. It may consist, however, in the extension of a thrombus from the right ventricle; but, in such case, it is of more recent formation, less firm, and rarely fills the vessel. Coagulation from stasis soon takes place in the branches of the vessel, and the original clot, thus extended by thrombosis, assumes a branched appearance corresponding to the ramifications of the pulmonary artery.

When the main trunk of the vessel is completely blocked,

\* See cases of aortic and mitral valve disease, *passim*.

† *Vide* an exhaustive paper on this subject, by Dr. G. Yeo in *The Dublin Journal of Medical Science*, for March, 1875.

death is very rapid; indeed, almost instantaneous. The patient is seized with a feeling of suffocation, is greatly agitated, and cries out for air; the chest heaves, and air freely enters the lungs, but the blood is not aerated; the right side of the heart is surcharged and paralysed by over distention, whilst the left side is empty and spasmodically contracted. Hence, death is caused partly by asphyxia and partly by syncope. The veins are turgid, the surface of the body is livid, and bloody foam usually issues from the mouth of the corpse.

Doctor Ormerod has recorded the case of a girl, aged twenty, paraplegic, and long bedridden, who complained of a queer feeling in her throat; cried out, at intervals, "Oh! my breath," threw her arms about wildly, and died within twenty minutes from the time she first complained of illness.\*

When the pulmonary artery is but partially occluded by a clot, the symptoms are less urgent, and less rapidly fatal; they closely resemble those of the collapse of cholera, as remarked by Dr. A. Carpenter.† Treatment can be of little avail in these cases. In examples of the more urgent form of the affection, no time is allowed for treatment of any kind; whilst in cases less rapidly fatal, the administration of alkalies, as previously mentioned (p. 1022), affords the only chance of alleviation. Venesection may assist in relieving distention of the right chambers of the heart, but it should be practised with caution, lest it favour coagulation by further enfeebling the heart. The two following cases of sudden death by pulmonary embolism have come under my notice.

CASE CXXIV.—*History of Great Privation; Anæmia, Stupor, and Debility; Swelling of the Tongue and Gums, without Salivation or Fætor; Leucocythæmia; Temporary Improvement; Sudden Death. Autopsy: Enlargement of Spleen; Embolism of Pulmonary Artery, and General Thrombosis of its Branches in both Lungs.*

George M., aged twenty-two years, was received into hospital, November 1st, 1865. He was a young man of some education,

\* *London Medical Gazette*, vol. ix., p. 788.

† *Lancet*, September 23rd, 1871.

who, owing to family reverses, had passed through the several social grades of apothecary's assistant, seaman on board a whaler, and fisherman on board a smack. He had left the latter employment three weeks previously, and, owing to some cause which he declined to mention, was forced to seek shelter in an out-house at Howth, where, during the interval of three weeks, he lay on a heap of straw, almost without food or drink.

When admitted, he was remarkably pallid, with a sallow tint, very drowsy, but easily roused, and then quite collected, answering questions clearly. The body was plump, but soft and flabby; surface and extremities cold; pulse weak, regular, and 90 in the minute. The stomach was extremely irritable, retaining neither food nor medicine, the vomited matter being of a bilious character. Despite the most careful examination, I failed to detect any organic disease whatever. The treatment consisted in sedatives, wine in moderate quantity, and liquid food.

A few days before his death, the tongue, which had been previously pale and moist, became so swollen and so tender, that he could not protrude it, but there was no salivation or fœtor of breath, and he had not, as he averred, taken a particle of mercury. A drop of blood taken from a puncture in his finger, and examined microscopically, seemed like water stained with blood, the margin of the field of vision being perfectly limpid and uncoloured; the white corpuscles were greatly in excess of the normal proportion, and nearly equal to the red in number.

His condition seemed to improve, sickness of stomach had ceased, he took more food, and slept better.

On the 17th of November, after returning to bed from the water-closet, he was observed to become suddenly faint, and he died in the course of a few minutes.

The body was sent for dissection to the School of Medicine, Cecilia-street, and was there examined. There was a good deal of subcutaneous fat. The organs were all sound, with the exception of the spleen, which was three times the normal size. A cylinder of nearly uncoloured fibrin had passed from the right ventricle into the pulmonary artery, which it entirely filled; the branches of this vessel, throughout both lungs, were likewise occupied, but not completely filled, by firm clot of a darker colour,

which, on slitting up the vessels, was removed entire, and found to present an arborescent form. The effort involved in the visit to the water-closet was obviously the cause of dislodgment of the clot from the right ventricle; but of its previous existence, no evidence was afforded by symptoms.

CASE CXXV.—*Emphysema of the Lungs with Bronchitis; Pallor; Musical Note of Systolic Rhythm at Lower Sternum; Sudden Death by Syncope. Autopsy: Heart Enlarged and Fatty; Right Chambers Thrombosed, and Tricuspid Valve Looped up by a Band of Fibrin; Pulmonary Artery and its Primary and Secondary Branches Plugged; Congestion of Bases of Lungs; Slight Thickening of Roots of Aortic Valves.*

Thomas H., aged seventy years, a labourer, was admitted into hospital, March 27th, 1871. For many years previous he had been in delicate health, subject to cough and dyspnoea.

At the date of admittance, he was pale and sallow; breathing somewhat embarrassed; pulse soft, regular, and 96 in the minute; cough, with frothy mucous expectoration. Physical examination elicited evidence of pulmonary emphysema, with bronchitis. Over the lower part of the sternum, a musical note was occasionally heard to accompany the first sound of the heart. A blister was applied to the chest, a stimulant expectorant was prescribed, and a few ounces of whiskey were allowed daily.

On the following morning (28th) he made a good breakfast, seemed in his usual health, spoke to the patients who occupied the adjacent beds, and did not complain of any special feeling of illness. Within ten minutes of the time when he thus addressed the other patients, he was heard to give a slight moan, and a few minutes afterwards, was found by the Sister on duty in the ward, lying across the bed, and all but lifeless; in the course of a few minutes more he died without a struggle.

The resident pupil on duty, the late Mr. Curran, who witnessed his death, reported to me, that during the last moments of his life the man was pale, and that death occurred apparently by syncope.

On examination of the body, the lungs were found to be em-



physematous ; the bronchial glands were enlarged, of dark colour, and cartilaginous density. The bases of the lungs were congested, and the bronchial membrane throughout was of a morone tint. The primary and secondary branches of the pulmonary artery were nearly filled with dark brittle coagulum.

The heart was somewhat enlarged, fatty on the surface, and exhibited a large "milk spot" on the anterior surface of the right ventricle. A mass of nearly decolorized fibrin, of the size and figure of a filbert, occupied the right auricular appendix, whence a thick band extended into the ventricle, looping up, in its passage, the anterior segment of the tricuspid valve, and establishing a connexion with a net-work of dense yellow fibrin which was entangled amongst the fleshy columns of the right ventricle. A large dark clot occupied the pulmonary artery, and extended, as above stated, into its primary and secondary branches.

The left ventricle was thick and somewhat dilated, and contained a mass of dark clotted blood. Both the mitral and the aortic valves were competent, but the roots of the latter, at their junction, were hard and spiculated in two situations.

The structure of the heart was in the granular stage of fatty degeneration. The existence of a musical note, accompanying the first sound at the lower part of the sternum on the 27th, may be accepted as showing that, even then, coagula had been already formed on the right side of the heart. The clot in the pulmonary artery was of a darker colour than that in the ventricle ; nevertheless, it is more than probable that it had been formed in the ventricle, whence it was suddenly dislodged, and impacted in the pulmonary artery. This hypothesis can alone supply a rational explanation of the sudden death. Dark coagula are not unusual in connexion with retiform thrombosis in the right chambers of the heart ; they are of more recent formation, less dense, and more loosely connected with the walls of the ventricle ; hence they are more readily detached.

## CHAPTER IX.

### NEUROSES OF THE HEART.

NEUROSIS, a term by which is implied derangement of innervation without structural disease of the heart, may consist in excessive or defective nervous activity of the organ. To the former category belong hysterical, dyspeptic, and gouty palpitation; and to the latter, palpitation from anæmia, chlorosis, and nervous exhaustion, cardiac asthma, angina pectoris, and exophthalmic goitre.\* Hysterical palpitation requires no special notice; it is characterized by the usual hysterical accompaniments. Palpitation arising from latent gout, or dependent upon the gouty diathesis, is characterized by flatulence, acidity of stomach, and the presence of lithates in the urine; it is amenable to remedies usually successful in the treatment of developed gout.

Case 126 (p. 1037) affords a good example of this form of the affection, and shows the benefit derivable from the treatment just indicated. The palpitation of anæmia and of chlorosis is readily distinguished by its associations, by which, likewise, an appropriate line of treatment is indicated. The derangement of the heart's rhythm, due to nervous exhaustion, and implied in the designation "irritable heart,"† includes not only palpitation from trivial excitement, physical or moral, but likewise sudden and extreme variations in the rate of the heart's action from purely emotional causes. Onanism, and the excessive use of tea and tobacco, are the ordinary causes of rhythmical derangement of this kind. Da Costa, however, has witnessed it, in numerous instances, in the persons of soldiers long exposed to anxious and fatiguing duty in the field during the late American war. In all such cases the left ventricle is in some degree dilated, from the twofold cause of

\* Palpitation has been already referred to at p. 161.

† *Toner Lectures* (No. 3), by J. M. Da Costa, Washington, 1874; and *Irish Hospital Gazette*, December 15th, 1874.

malnutrition and over-action, and, not unfrequently, a systolic murmur of adynamic origin exists at the apex.\*

The following example of neurosis, from the frequent use of strong tea, has come under my notice. Two brothers, engaged in the tea trade, whose duty as "tea-tasters" required them every day to sip strong infusions of different samples of the article, became pallid and nervous; the pulse became rapid under the most trivial excitement; the heart beat tumultuously; there was dizziness, and occasional inability to maintain the erect posture without support, accompanied by a tendency to fall backwards when the head was raised. Under a short course of treatment with quinine and strychnine, whilst giving up the use of tea at their meals, and carefully avoiding to swallow any portion whilst tasting it in their daily avocations, these gentlemen were quickly relieved from the symptoms above described.

The discontinuance of the habit of onanism, the use of bromide of potassium thrice daily, in doses of grs. xv, and cold showers, rarely fail to arrest the palpitation from sexual abuse, and if the disease be not inveterate, to effect a rapid cure. Iron and quinine are sometimes required in the after-treatment of such cases; these agents are especially indicated where an adynamic murmur exists at the apex of the heart.

I recently witnessed, in the person of a young man, aged nineteen, a remarkable example of functional derangement of the heart from nervous exhaustion and excitement combined. He had been reading for a competitive examination, and had, within a few hours previously, received the intelligence of his having obtained the first place. When he called on me for the purpose of obtaining a certificate of health for the Civil Service, he was returning home from the school where he had been educated, after announcing his success, and receiving an ovation from his fellow-students. He was pale and fidgety; the pulse was 150, and intermittent; the impulse of the heart was strong and abrupt, and accompanied at the apex by a very distinct thrill, whilst the sounds were sharp and loud. I directed him to return in the course of the day, after he had taken a walk and some refresh-

\* See pp. 282 and 568.

ment. On his second visit, two hours later, the pulse was 80 and free from intermissions; the action and sounds of the heart were normal, and fremitus no longer existed.

Professor Laycock thinks that extreme nervous excitability of the heart, such as that exhibited in the preceding case, is premonitory of organic disease;\* I cannot agree with him in this opinion.

The subjoined tracing, though not exhibiting irregularity, shows the character of the pulse in some cases of irritable heart.

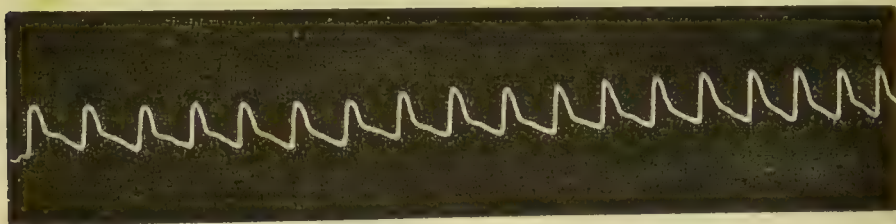


FIG. LXVII

Irritable heart, with anæmia (Meehan).

Tracing taken June, 1873.

*Cardiac asthma* is a peculiar form of dyspnoea, of an intermittent character, and dependent upon organic, and most frequently, valvular disease of the heart. It is characterized by restlessness and agitation, craving for air, quick and deep respiration, congestion of the face, palpitation, and increased rapidity of pulse. In those who are subject to it, the paroxysm is readily induced by physical or mental excitement. It depends upon temporary derangement of balance between the circulatory and respiratory functions, consequent upon instability of cardiac innervation. I have most frequently witnessed it in connexion with disease of the aortic valves and aorta.†

In the following case, however, there was no valvular disease, whilst undeveloped gout was obviously the cause of the paroxysmal seizures.

\* *Dublin Journal of Medical Science*, July, 1873.

† See Case 80, p. 858; Case 87, p. 866; Case 101, p. 889; Case 102, p. 918; and Case 106, p. 927.



CASE CXXVI.—*Paroxysms of Dyspnœa, with Palpitation and Flatulence, in Connexion with Undeveloped Gout. Recovery.*

Mr. R., aged fifty years, superintendent of a large mercantile establishment, consulted me on the 12th of January, 1870. Three years previously he had experienced his first symptoms, in Paris. He was anxious looking, haggard, and somewhat excited by his visit to me. Pulse 102, and weak but regular; apex-beat in nipple-line; sounds, especially the first, rather clear, but unaccompanied by thrill or murmur. Family gouty; has been himself repeatedly threatened with gout on taking malt drinks; urine deposits a lateritious sediment. He is occasionally, towards morning, suddenly awakened from sleep by a feeling of great oppression of the chest, with palpitation, which obliges him to start up, raise his arms, and struggle for breath. The paroxysm lasts about five minutes, after which he has complete relief. Has been taking brandy rather freely to relieve these symptoms. Moderate exercise agrees with him; he is subject to flatulence, and is relieved by eructation; bowels regular. To have light nutritious diet, and a moderate allowance of claret, but no brandy; and at night, a draught containing ℥xx of liquor of the hydrochlorate of morphia, with ℥ij of hydrocyanic acid, and ℥x of chlorodyne, in an ounce of water.

On the night of the 16th he had the premonitory sensation of a fit, which he managed to ward off by "shaking himself up." This feeling recurred five times in succession on going to sleep, and on each occasion he succeeded in averting a fit by the manœuvre just described. He ultimately fell asleep after taking some brandy and solid food.

17th. Pulse full, regular, 96; urine clouded, and acid. To have a belladonna plaster to precordium, and every morning a pill composed of aromatic powder and mercurial pill, of each grs. ij, with gr. j of the acid extract of colchicum. A draught, consisting of tincture of Indian hemp, ℥xv, hydrocyanic acid, ℥ij, and chloric ether, ℥x in an ounce of water, to be taken at night, if necessary; and Vichy water (L'Hôpital) in the course of the day.

21st. Pills act twice daily; urine now clear; has had only

one slight paroxysm since last report. To continue pills and Vichy water.

February 2nd. Much improved. No recurrence of dyspnœa or palpitation. Pulse 72, regular. Urine clear. Slight muscular pains in thighs and upper arms ; but, with this exception, he regards himself as well.

The group of symptoms implied by the designation *angina pectoris* has been long familiar to clinical observers, at least in general outline. Typical examples may be found recorded by Morgagni.\* Heberden, however, was the first who employed the name “angina pectoris,” and gave a special individuality to the symptoms included under it, by presenting them in a collective form, and connecting them, theoretically, with a particular morbid condition which he designated “spasm” (distention).† He does not indicate precisely the seat of assumed “spasm,” which, a few years later, was localized in the heart by Macbride.‡ Shortly after the publication of Heberden’s memoir, an anonymous correspondent, who identified the disease in his own person, by means of the description given of the symptoms by Heberden, presented him with a graphic sketch of his own case, dwelling especially upon the substernal pain, shooting across the chest and down the arms as far as the elbows, and the accompanying dyspnœa, during the paroxysm. He bequeathed his body to Heberden for examination after his death, which took place in one of these attacks ; the body was examined by John Hunter. The only lesion discovered was slight ossification at the root of the aorta.

Dr. Fothergill’s paper was, after Heberden’s, the next contribution on the subject.§

Doctor Wall, of Worcester, communicated to Heberden the particulars of a case of *angina pectoris* which had been under his observation. After death, the heart was found to be fatty,

\* *De Sedibus et Causis Morborum*, liber ii.

† *Medical Transactions*, No. VI., vol. ii., July, 1768. See also *Commentaries on the History and Cause of Diseases*, 1802.

‡ *Medical Observations and Inquiries*, vol. vi., 1778.

§ *Ibid.*, vol. v., 1773.

the aorta was dilated and atheromatous, and the aortic valves were ossified.\*

Doctor Butter published a special work on this subject,† in which he endeavoured to show that angina pectoris is a manifestation of gout, often ceasing entirely with a fit of that disease.

Eight years later, Dr. Parry's well known treatise appeared,‡ and contained the best exposition of the subject presented up to that date. Dr. Parry, having reference to the two most prominent symptoms of the affection, namely, faintness, and painful constriction of the chest, named it *syncope anginosa*. He adopted the view first propounded by the celebrated Edward Jenner, of the dependence of angina pectoris upon ossification of the coronary arteries, regarding calcareous change with dilatation of the aorta, and enlargement of the heart, as subordinate causes, and attributing the attendant pain to accumulation of blood in the heart and large vessels. He admitted that gouty patients are the most liable to attack, but considered that organic disease of the heart, so commonly associated with chronic gout, was the cause of this proclivity.

It is unnecessary to enumerate the several memoirs which have been since written to illustrate the subject of angina pectoris. Many of these will be necessarily referred to in the sequel.

M. Jaccoud§ divides angina pectoris into true and false, after Lartigue. The true form, according to him, may be idiopathic or primary, or it may be symptomatic. It may be doubted, however, that angina pectoris is ever strictly primary, except in the few instances in which it is the only actual indication of gout. Trousseau, indeed, held that in many cases it is nothing else than "partial epilepsy," or "epileptic neuralgia."|| But few, I apprehend, will adopt this view. He admitted, also, that it may arise from gout or rheumatism.

In the great majority of examples of angina pectoris, organic

\* *Medical Transactions*, vol. iii., 1785.

† *A Treatise on the Disease commonly called Angina Pectoris*, 1791.

‡ *An Inquiry into the Symptoms and Causes of the Syncope Anginosa*, London, 1799.

§ *Dictionnaire de Médecine et de Chirurgie*, vol. ii.

|| *Clinical Medicine*, Sydenham Society's Edition, vol. i., p. 65.

disease of the heart is clearly revealed by physical evidence, and in a still larger percentage of cases, *post mortem* examination would demonstrate its presence. Valvular disease, which, in cases of angina pectoris, is most frequently aortic, and likewise dilatation and atheromatous change of the aorta above the valves, may be detected by physical examination. Not so, however, in some instances, fatty degeneration of the heart; but the symptoms indicative of this condition are usually so well pronounced in the exceptional cases, as to supplement the want of physical signs, and leave very little doubt as to the existence of fatty change of the heart.

Aneurism of the aorta has been occasionally met with in connexion with angina pectoris; but, as most cases of aneurism have not been associated with angina pectoris, and as the latter is by no means a characteristic sign of the former disease, the association must be regarded as a coincidence. Calcification of the aorta and of the coronary arteries, and fatty degeneration of the heart, so frequently witnessed in connexion both with aneurism and angina pectoris, may serve to explain the coincidence. It may be useful here, as exhibiting the unsettled state of opinion with reference to the pathogeny of angina pectoris, to summarize the views held as to its cause.

- (a) Spasm (distention); Heberden.
- (b) Spasm of the heart; Macbride, Latham.
- (c) Calcification of the coronary arteries; Jenner, Parry.
- (d) Spasm of the heart, and pressure of the cardiac nerves against calcified coronary arteries; Sir Everard Home.
- (e) Neuralgia located in the pneumogastric, sympathetic, and brachial nerves; Desportes, Laennec, Piorry, Flint, Jacoud.
- (f) Nervous and spasmodic affection arising from gout; Butter, MacQueen, Johnston, and Blackall.
- (g) Hyperæsthesia of the cardiac plexus; Romberg.\*
- (h) Hypercinesia, with hyperæsthesia; Bamberger.†

It is, I think, essentially a neurosis; engaging primarily, in most instances, the cardiac plexus, whence it is reflected through

\* On the authority of Niemeyer.

† *Ibid.*



the sympathetic to the spinal nerves, and occasionally through the pneumogastric to the cesophageal plexus. It is, in short, a cardialgia of the most aggravated character, which may or may not be associated with organic disease of the heart. But in the great majority of cases, it is so associated. Of 19 cases of angina pectoris collected from the *Transactions of the Pathological Society of London*, by Dr. J. W. Ogle,\* the coronary arteries were calcified in 12; the heart was fatty in 12; the aorta was atheromatous in 10, and valvular disease existed in 6 cases. Grave structural changes of the aortic and cardiac plexuses have been found by Lancereaux.†

The occurrence of death during a paroxysm must be attributed to syncope from excessive pain in a vital organ, weakened by disease. Out of 13 cases of angina pectoris reported by Dr. Ogle, in which *post mortem*‡ examination showed the existence of atheroma of the aorta, with partial or complete occlusion of one or both coronary arteries by calcareous matter, or by fibrin, sudden death occurred in 11 instances, and in 3 of these the heart was in a state of fatty degeneration. Hence, I regard the designation "syncope anginosa,"§ as most appropriate.

I cannot look upon angina pectoris as necessarily fatal. In the primary form it certainly should not be so regarded; but the cases in which it is primary are comparatively few. The mortality of the disease is due rather to its usual associations, organic disease of the heart or aorta, than to its essential gravity. Flint has reported several cases in which, after repeated attacks, the patients were exempt from the affection for many years.|| I have met with a few cases of the same kind. Case 44, p. 647, and Case 93, p. 876, may be referred to as examples.

The immediate cause of the paroxysms is, most frequently, quick walking soon after meals, the ascent of an acclivity, movement against the wind, or emotional excitement, especially

\* *British and Foreign Medico-Chirurgical Review*, No. 92, October, 1870, p. 449.

† *Gazette Médicale*, 1867, p. 432.

‡ *Loco citat.*

§ Parry.

|| *Diseases of the Heart*, second edition, 1870.

anger. The patient then suddenly feels an excruciating pain of a constrictive character shooting across the chest at the level of the middle or lower portion of the sternum, and extending down one or both arms, most frequently the left; it not unfrequently radiates to the shoulders and back of the neck, round the chest in the course of the thoracic and intercostal nerves, and over the abdomen and groins, following the course of the branches of the lumbar plexus. The sufferer is forced to stand still and seek support, has a feeling of impending death, looks pale, haggard, and greatly alarmed, and fears to breathe lest he aggravate his pain. The pulse usually maintains its normal rate and volume, but exhibits occasional intermissions; it may, however, be very slow or very rapid during the paroxysm. There is no palpitation, and air freely enters the lungs when the patient essays to breathe. After a period of a few seconds, or a few minutes, the paroxysm ends quite as suddenly as it commenced, and generally with eructation of flatus. The patient is then in his normal condition, but is puzzled as to the nature of his attack, and alarmed at the prospect of its recurrence. I have known spasm of the cesophagus, as indicated by globus, to characterize the earlier invasions of the disease. Sighing is usually a concomitant symptom, whilst the posture assumed by the patient may vary from the erect to the bent. The intervals between these seizures are of uncertain length, and depend, in a great degree, upon the habits and temperament of the patient. At first they are usually considerable, varying from days to months, or even years. I have known a gentleman to suffer only one attack, now many years ago. He is at present in excellent health; but in his case gout, of which he subsequently had repeated fits, was the only assignable cause, and his immunity since that date is due to his active habits and caution as to food and drink.\*

Unfortunately, however, the fits usually recur with progressively increasing frequency; they are likewise more severe and protracted in the progress of the case; and towards its termination several may occur within the twenty-four hours, being provoked by the slightest movement or chill, even that involved in getting into or out of bed.

\* See Case 128.

The disease may be fatal within a fortnight after the first seizure, or even in the first attack.\* The celebrated Dr. Arnold, of Rugby, died in less than three hours from a primary invasion of angina pectoris.†

Genuine angina pectoris may be distinguished from the less grave affections, cardiac and spasmodic asthma, by the presence of the characteristic substernal pain, and by the absence of quick, laboured, and sonorous respiration, craving for air, agitation of the body, palpitation, and rapidity of pulse. In the cardiac form, asthma is further characterized by engorgement of the cervical veins, congestion of the face, and by the signs of distention of the right side of the heart. Laennec obviously failed to draw this distinction, hence the less grave aspect which angina pectoris presented to him.

According to Pinel, angina pectoris is more common in winter than in summer;‡ a difference which is probably due to the unfavourable effect of cold upon the surface of the body. It would seem that England enjoys an unenviable distinction above all other countries by the prevalence of the disease amongst its population. The rich are certainly more liable than the poor, and preeminently those of high intellect and anxious temperament. The male is more frequently affected than the female sex. Of 88 cases collected by Sir John Forbes, only 8 occurred amongst females.§ The age of the persons attacked is rarely under forty-five.

Angina pectoris is a comparatively rare affection. I have met with only 6 genuine examples. In 3 of these the disease was connected with latent gout, in 2 with fatty disease of the heart, and there was no organic lesion in one instance. As to the association of angina pectoris with valvular disease, I will only remark, that out of a total of 136 cases of valvular lesion recorded in my notes, angina pectoris was present in 3 only.

Of 188 cases of organic disease of the heart, noted from personal observation by Flint, only 8 exhibited angina pectoris.

\* Latham on the *Diseases of the Heart*, vol. ii., Lectures xxxvii., xxxviii.

† See his life, by Dean Stanley.

‡ Jaccoud, *loco citat.*

§ *Cyclopaedia of Practical Medicine.*



The prognosis is eminently grave, even in the most favourable cases, owing to the possibility of latent organic disease of the heart or aorta; but where undeveloped gout is, presumably, the only cause in operation, a less unfavourable prognosis may be given. Where organic disease of the heart or the aorta manifestly exists, no hope of recovery can be entertained, although life may, even in such cases, be prolonged for years.

*The treatment* of the disease resolves itself into that which is proper during the paroxysm, and that which should be pursued in the intervals with a view to averting its recurrence.

The treatment of a patient in a paroxysm of angina pectoris should be regulated by the most urgent symptoms present. If, in any degree, venous turgescence were exhibited, I would not hesitate to draw blood by venesection, or by cupping at the precordium, to the amount of four or five ounces. I should expect that a weak and intermittent pulse, associated with superficial congestion, would expand and become steady under partial abstraction of blood, owing to the relief thereby given to the distended right ventricle. Both Trousseau and Jaccoud object to blood-letting in this disease under any circumstances, but, as I believe, on insufficient grounds. Where pallor without congestion characterizes the superficial surface, as is usually the case, dry-cupping over the heart, followed by a sinapism or a stimulant embrocation, would be more appropriate. If pain were the prominent symptom, opium with ether should be given at the same time, 3ss to 3j of Hoffman's anodyne with ℥xxx of the liquid extract of opium. Sulphuric ether, given by inhalation, to the amount of 3iij to 3iv, might be substituted for the preceding, or, better still, the nitrite of amyl, from ℥v to ℥x, which, in many cases, promptly arrests the paroxysm.\* The inhalation of nitrite of amyl is not, however, free from danger, and should be administered with great caution, especially where organic disease of the heart or aorta is known to exist. Dr. Haddon mentions two cases in which the most alarming symptoms,

\* *Vide* a most interesting report of his own case by Dr. Herries Madden, *Practitioner*, December, 1872; see also Lauder Brunton, *Lancet*, July 27th, 1867; Leishman, *Practitioner*, September, 1869; Wilks, *Lancet*, January 16th, 1869; H. C. Wood, *American Journal of the Medical Sciences*, October, 1871; and Haddon, *Edinburgh Medical Journal*, July, 1870.



including stoppage of the heart's action and general congestion, followed its use. In view, however, of the not improbable existence of fatty disease of the heart, it would be in the highest degree imprudent to venture upon the administration of chloroform. I cannot agree in the opinion expressed by Niemeyer, that opiates and other narcotics are to be avoided.\* On the contrary, the hypodermic injection of morphia, or of atropia, is often followed by great relief.

Where faintness is the principal symptom exhibited, brandy should be freely given till reaction has been induced. Warmth should be, at the same time, applied to the feet, and the best form in which to apply it is that of a warm mustard foot bath. In the last mentioned cases, viz., where great debility is the chief characteristic of the paroxysm, Duchenne and Aran have successfully practised electrization of the left side of the thorax. Both these physicians have reported examples of apparently complete cure effected by electricity so applied.† Eulenburg, of Berlin, has likewise had some success with electricity; he prefers the constant current (up to 30 elements of Siemen's), the positive pole being applied to the sternum by means of a broad plate, and the negative in the situation of the sympathetic chains in the neck. The cases best adapted for the use of electricity are, he thinks, those which exhibit irritation of the excito-motor or the vaso-motor centre, as shown by small and hard pulse, and more or less excitement of the heart.‡

In the treatment of the patient during the interval between the fits, the physician should be guided by the conclusion at which he may have arrived, as to the cause of the affection in the case before him. If it were manifestly latent gout, the appropriate remedies in the treatment of that disease, and preeminently the salts of lithia, with a suitable diet and regimen, should be prescribed, whether organic disease existed or not. Case 128, p. 1049, affords a good example of the beneficial action of lithia and regulated regimen in the treatment of angina pectoris of gouty origin. Chloral hydrate given every

\* *Text-Book of Practical Medicine*, vol. i., p. 371.

† See Trousseau, *Clinical Medicine*, vol. i.

‡ *Medical Times and Gazette*, May 7th, 1870.

night, in doses of grs. xxv to xxx, in combination with sulphuric ether and aromatic spirit of ammonia, has been found by Dr. Strange to give great relief. After the continuous use of the drug for four months, an additional half dose, every night, became necessary.\* Fothergill has mentioned a case in which, under the use of the Bath waters, an apparently perfect cure was effected, the patient having had no fit for a period of twenty years.† Trousseau recommends Fowler's solution, and also the simultaneous use of bicarbonate of soda and belladonna, administered according to the method of Bretonneau; viz., in doses gradually and alternately increasing and diminishing at intervals, for a protracted period.‡ Anstie also strongly recommends Fowler's solution, in doses of ℥v, thrice daily; and where the stomach will not tolerate this, he suggests the sub-cutaneous injection of strychnia, to the amount of the one-hundred and twentieth to the one-hundred and sixtieth of a grain.§

With a similar object in view, namely, that of arresting degeneration of tissue by promoting general nutrition, phosphorus may be administered in minute doses, one-fortieth to one-sixtieth of a grain twice daily, as recommended by Dr. Broadbent.|| I have no personal experience of this agent in the treatment of angina pectoris; but, encouraged by the results of Dr. Broadbent's experience, I would certainly give it a trial in suitable cases.¶

Doctor Rufus K. Hinton recommends the use of bromide of ammonium as a prophylactic in this disease. He publishes two cases of indubitable angina pectoris, in which, given in doses of grs. xv to xx, the bromide of ammonium would seem to have been efficacious in preventing the paroxysms.\*

In addition to the examples of angina pectoris incidentally

\* *Medical Times and Gazette*, September 4th, 1870.

† *Loco citat.*

‡ Trousseau, *opere citat.*

§ *British Medical Journal*, November 11th, 1871.

|| *Practitioner*, January, 1875.

¶ For a full *résumé* of the subject of angina pectoris, especially in reference to treatment, see *The Boston Medical and Surgical Journal* of May 21st, 1874.

\*\* *Philadelphia Medical and Surgical Reporter*, March 6th, 1875.

recorded in the preceding pages (Case 44, p. 647; Case 47, p. 681; and Case 101, p. 889), the two following cases may be given here.

CASE CXXVII.—*Angina Pectoris of the Ingravescens Character; Double Aortic Murmur; Improvement under the Use of Lithia and Chloral Hydrate; Intercurrent Jaundice; Death by Exhaustion. Diagnosis: Hypertrophy of the Left Ventricle; Obstruction and Inadequacy of the Aortic Valves, and Dilatation and Atheroma of the Aorta.*

Mr. S., aged forty-two years, a hard-working barrister, of anxious temperament and regular habits, consulted me on the 2nd of July, 1873. Had not had rheumatism or gout. Two years previously, he experienced, for the first time, pain across the front of his chest, and lasting about ten minutes, after exertion. He had another similar attack a few weeks afterwards. Latterly, these "spasms," as he termed them, had been more frequent. They occurred most frequently at night, but seldom in bed, and never, except after exertion of some kind. They had repeatedly occurred on getting out of bed in the morning. Exposure to cold, or the application of cold to the surface, such as washing with cold water, was certain to bring on a "spasm"; walking against the wind had a similar effect.

Some of these attacks had been very mild, consisting only of pain across the chest lasting for a few minutes, whilst others had been very severe, attended with excruciating pain in the chest, back of the neck, and down the left arm, to the back of the hand and fingers. These latter had been accompanied by strong and rapid pulsation of the heart and gasping for breath, obliging him to support himself against some fixed object. Food of certain kinds, such as pastry, which caused flatulence, rarely failed to bring on a fit.

He was remarkably anxious-looking and haggard; could sleep well, and usually on the left side. Pulse 84, full, soft, and regular, but collapsing, and visible after excitement. The apex of the heart pulsated feebly in the nipple-line; first sound at apex very faint; second sound replaced by a loud diastolic murmur, transmitted from the base. This latter murmur was audi-



ble over the entire front of the chest, but with greatest intensity at the lower portion of the sternum.

Over the base of the heart, a faint but rough systolic murmur was likewise heard; it was transmitted through the arch of the aorta, and into the carotids. The action of the bowels and kidneys was regular, but the urine occasionally exhibited a deposit of lithates. An opiate plaster was applied to the precordium; he was directed to apply a few leeches at midsternum, or to be dry-cupped in that situation, in the event of a paroxysm of pain occurring; also to take, thrice daily, ℞ijj liquor. strych., (B. P.), with ℞ijj tinct. digitalis, and grs. iss sulph. quiniæ, in solution.

On the 28th of October he was slightly jaundiced, and the liver was congested and tender; grs. vj of the citrate of lithia were prescribed daily, and likewise an aperient pill containing inspissated ox gall. A draught, containing grs. xx of chloral hydrate was directed to be taken at night should a paroxysm occur.

On the 26th of December, he reported himself as greatly improved. Since last visit he had been rarely attacked with pain in the chest or arm; could sleep through the night, and on two occasions when the "spasm" came on in bed, he was able to control it by means of the chloral draught. On the preceding day he had walked ten miles without an attack, or feeling fatigued. Had cough, with expectoration of viscid mucus, and suffered also from flatulence. For the former, ℞xxx of compound tincture of camphor, and of syrup of cinchona, with ℞viij of cherry-laurel water were prescribed, and, for the latter, Belloc's charcoal.

In March, 1874, the paroxysms became more frequent and more severe; the pain then radiated from the epigastrium to the back and over the abdomen; the jaundice returned, the appetite failed, and he could not obtain sleep. He wasted considerably. Cough was more urgent, and was attended with slight hæmoptysis. The face assumed an anxious and haggard expression; the feet became swollen; oedema of the lungs set in; he could rarely assume the recumbent posture, even for a moment, owing to extreme dyspnœa, and died, worn out, on the 13th of May. The body was not examined.



CASE CXXVIII.—*Latent Gout ; Angina Pectoris accompanied by Intermissions of the Action of the Heart ; Slow and Suspicious Breathing during Sleep ; Nephritic Colic ; Articular Gout ; Recovery under the Use of Lithia and Alkalies.*

A professional man, in good business and of active habits, aged about forty-five years, naturally florid, but latterly become fat and pale, consulted me in January, 1868, in reference to palpitation, accompanied by intermissions of the cardiac impulse and of the radial pulse, which he latterly began to experience on going up stairs, or being otherwise hurried in his movements ; he had also felt, on these occasions, a dull tensive pain behind the sternum. He further informed me that, during sleep, his breathing had been noticed, of late, to be occasionally slow, sighing, and oppressed. Pulse 86, and regular ; appetite indifferent ; has had flying gouty pains in the feet and in the muscles of the chest. Lithia was prescribed, and warm mustard foot baths were directed, with a view to develope an attack of gout in the feet.

I heard nothing further of this gentleman's health till February, 1870. He had been fairly well during the interim, and continued to discharge his professional duties, which were laborious, without interruption. He had, however, been much annoyed by acidity of stomach, and had noticed, from time to time, a deposit of lithates in the urine.

On the night preceding my visit to him he had a severe attack of nephritic colic, after having dined heartily upon salmon. He was quickly relieved, under active treatment by means of counter-irritants and alkaline diuretics, and, in the course of a couple of days, was able to resume his professional duties.

January, 1875. Since last report he has had repeated attacks of gout in the feet, and, for some time after each of these attacks, he has felt much better in regard to his general health. No return of angina, from which he has been now free for more than seven years. Habits most carefully regulated in regard to diet and exercise.

The complex morbid condition known under the designation of

*Exophthalmic Goitre*, *Graves' disease*, or *Basedow's disease*, which has been already referred to at p. 489, consists in its complete form, of the "symptomatic triad," palpitation with throbbing of the arteries of the neck, vascular turgescence and enlargement of the thyroid body, and prominence of the eyeballs, was first described by Graves in his clinical lectures delivered in 1835. Flajani, in 1802, had published three cases, now regarded as belonging to this category, amongst the symptoms of which, however, exophthalmia was not included.\* Galezowski† claims for Demours the original description of the disease in 1818. Parry, in 1825, recorded seven cases of thyroid enlargement in connexion with affections of the heart.‡ According to Hirsch, Basedow was the first writer who described the disease; he proposes therefore to name it after him; Basedow's account of the affection was, however, not published till 1840.§ Jaccoud has shown|| that to Graves is due the merit of having been the first to direct attention to this singular affection as a distinct morbid entity.

According to Dr. Stokes, the essential features of the affection are the following :

1. Increased force and rapidity of the heart's action, of long continuance, and without fever.
2. Excited action of the carotid and thyroid arteries.
3. Enlargement of the thyroid gland, varying with the force of the heart.
4. Enlargement of the eyeballs, without any disease of the orbits or brain.

He regards it as "a special form of cardiac neurosis, which may result in organic disease," and, probably, a similar affection of the cervical vessels.¶

Niemeyer's view of the pathology of the disease is virtually

\* *Collezione d'Osservazioni*, tom. iii., p. 270.

† *Gazette des Hôpitaux*, 1871, p. 425.

‡ Collections from the unpublished writings of the late Caleb H. Parry, M.D., London.

§ Casper's *Wochenschrift*, March, 1840.

|| *Leçons de Clinique Médicale de R. J. Graves*, ouvrage traduit et annoté par le Docteur Jaccoud, 1862, tom. ii., p. 290.

¶ *The Diseases of the Heart and Aorta*, p. 279.

identical with that of Stokes. He attributes the affection to palsy of the vaso-motor nerves, which, in his opinion, "fully accounts for the dilatation and increased pulsation of the carotid and thyroid arteries, as well as for the œdematous swelling of the thyroid gland and intra-orbital fat." There is likewise, he thinks, "a sub-paralytic state of the vessels of the muscles of the heart."\*

Trousseau, while admitting that neurosis of the heart constitutes an essential element of the disease, does not regard it as the primary lesion. He looks upon the affection as "a neurosis of the sympathetic, from congestion or structural change of the ganglionic system." In one instance he found, on *post mortem* examination, the inferior cervical ganglia of the sympathetic, especially that of the right side, greatly enlarged, and highly vascular; its connective tissue hypertrophied, and exhibiting nuclei and fusiform cells, whilst the proper nerve constituents, both cellular and fibrous, were wasted. The cardiac plexus was simply vascular.† Drs. Cruise and R. M'Donnell found the inferior cervical ganglion almost entirely replaced by areolar and adipose tissue.‡ According to Dr. J. Begbie, the disease is essentially anæmic.§ Bellingham|| and Makenzie,¶ were of the same opinion, whilst Dr. J. W. Begbie regards anæmia as the primary condition, vaso-motor paresis being a direct consequence of this.\*\*

Doctor Lauder Brunton is of opinion that the palpitation, so characteristic of this disease, is due to direct stimulation of the accelerating nerves of the heart, which descend from the vaso-motor centre in the medulla oblongata in company with the vertebral artery, and, after passing through the inferior cervical ganglion of the sympathetic, are supplied to the heart. Irritation of this ganglion would, therefore, account for cardiac excitement.

The remittent engorgement of the thyroid vessels may, in

\* *Text-Book of Practical Medicine*, vol. i., p. 375.

† *Clinical Medicine*, Sydenham Society's Edition, vol. i., p. 575-82.

‡ See abstract of Case, p. 1061.

§ *Monthly Journal of Medical Science*, 1849, p. 495, and *Contributions to Practical Medicine*, p. 176.

|| *Diseases of the Heart*, p. 532.

¶ *Diseases of the Eye*, 1854.

\*\* *Edinburgh Medical Journal*, vol. ix, part i., p. 206.

his opinion, depend upon paralysis of their vaso-motor nerves which are derived from the second cervical ganglion, or upon "inhibition" of these through other nerves. He thinks that protrusion of the eyeballs is most probably due to turgescence of the blood-vessels and lymphatics in the orbit; remarking that, in the experiments of Bernard, exophthalmia was readily produced by irritation of the nerve-filaments connecting the first and second dorsal ganglia of the sympathetic with the spinal cord.

Finally, he calls attention to the loss of *consensus* between the movements of the eyeball and those of the upper eyelid, which was exhibited in one of his cases, and ventures to account for this symptom on the assumption of disturbed innervation of the lids, as propounded by von Gräfe; and especially of the upper lid, through the levator muscle, which derives its nerve supply in part from the sympathetic.\*

Mr. Swanzy has reported a case of exophthalmic goître, in which the want of consensual movement of the upper eyelid, when the patient looked down, was well pronounced; nictitation was likewise remarkably slow and infrequent in this case, both phenomena depending, in his opinion, upon deranged innervation of the lids.†

Whilst I believe that the singular combination of symptoms constituting exophthalmic goître is essentially dependent upon vaso-motor paresis, I feel with Dr. Cheadle that the frequent coexistence of dilatation of the pupils, a condition which indicates rather irritation than paralysis of the sympathetic nerves, with elevation of temperature, epistaxis, and diarrhoea, constitutes an objection which must be answered before this theory can be unreservedly adopted. Such an answer cannot at present be given.

Cardiac excitement is usually regarded as the primary symptom. Trousseau, however, recognizes a previous state of nervous irritability, manifested by peevishness and impatience. This is soon followed by a sense of fulness and throbbing in the head and eyes, palpitation, throbbing at the epigastrium, and often vomiting and diarrhoea. The patient now loses flesh, notwithstanding the existence of a morbid appetite, which, however, is

\* *St. Bartholomew's Hospital Reports*, vol. x., 1874.

† *Irish Hospital Gazette*, vol. i., p. 258.



soon replaced by anorexia. In females, derangement of the menstrual function is an early symptom, and, as noted by Dr. Cheadle, constant elevation of temperature and occasional profuse sweating and epistaxis.\* The other two cardinal symptoms now gradually declare themselves, enlargement of the thyroid usually preceding the exophthalmia. The order of the two latter symptoms may, however, be inverted, as it was in the majority of my cases; or either may be absent, most frequently the thyroid enlargement, constituting an example of the "incomplete" form of the disease.

Exophthalmic goître may be acute or chronic. Dr. Adams has known ocular prominence to be suddenly produced by a violent fit of coughing and retching,† and Dr. R. Taylor has witnessed its occurrence in the course of one night.‡ Trousseau has recorded the case of a female, sixty years of age, in whom the three prominent symptoms of the affection were developed in the course of a single night from excessive mental emotion.§ The disease is, however, most frequently of slow and progressive growth, the rate of its progress being unsteady and fitful. A fright or a mental shock of some kind is usually the starting point of the symptoms. Protracted watching and great anxiety may give rise to it. Stokes has known it to be produced in the male subject by long continued bleeding from hæmorrhoids.

The great majority of those who suffer from exophthalmic goître are females. Males are occasionally, though rarely, the subjects of it. I have not seen an example of it in the latter sex; but Stokes|| and Trousseau have each witnessed one example. Cheadle,¶ Moore,\*\* Foot,†† Begbie,‡‡ and Reith§§ have each re-

\* *St. George's Hospital Reports*, vol. vii., 1875, p. 81.

† Stokes on *Diseases of the Heart and Aorta*, p. 295.

‡ *Medical Times*, May, 1856, p. 516.

§ *Opus citat.*, p. 575.

|| For further report of this case by Dr. L. McDonnell, see *Dublin Journal of Medical Science*, vol. xxvii., p. 200.

¶ *St. George's Hospital Reports*, vol. iv.

\*\* *Dublin Quarterly Journal of Medical Science*, November, 1865, p. 344.

†† *Irish Hospital Gazette*, vol. ii., p. 179.

‡‡ *Dublin Hospital Gazette*, vol. ii., p. 107, quoted from Case Book of *The Edinburgh Medical and Surgical Journal*.

§§ *Medical Times and Gazette*, November 11th, 1865.

corded a case of the disease in the male sex. Of 50 cases collected by Withusen, the subjects were males in only 8 instances.\* The period of life at which it is most common is between puberty and the age of forty. From twenty to twenty-five years is, according to Trousseau, the age at which it usually occurs. Of my 6 cases, 3 were over twenty-five, and 2 were forty years of age; but, in the latter cases, the disease had been in existence six and eight years respectively when the patients came under my notice. Niemeyer remarks that men affected with this disease are usually advanced in life, whilst, in females, it generally appears in youth.†

Of the three characteristic symptoms of this disease, palpitation is usually the first exhibited. In Case 134, however, enlargement of the thyroid gland was the first symptom noticed; palpitation with further and rapid growth of the thyroid supervening six years later, on the occurrence of uterine hæmorrhage. The pulse is rarely under 120, whilst it not unfrequently attains to 160 in the minute. In cases uncomplicated by hypertrophy of the left ventricle it is small, and most frequently regular; but irregularity or intermittence of the pulse is occasionally witnessed in this, as in all other forms of cardiac neurosis.

The subjoined tracing, taken from a female suffering from exophthalmic goître in a typical form, the notes of whose case I have unfortunately lost, shows well the irregularity and intermissions which characterize the pulse in many cases of the affection.

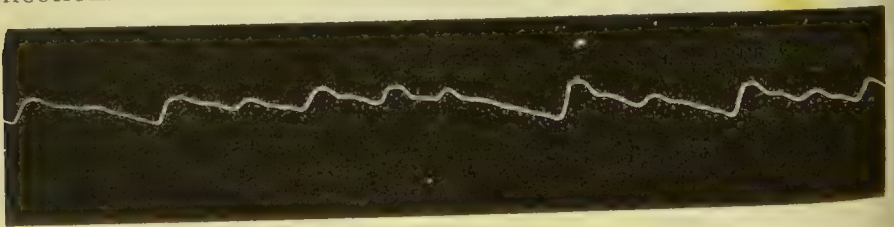


FIG. LXVIII

Exophthalmic Goître. (McG.)

Pulse 70. September 17th, 1874.

The impulse of the heart is abrupt and energetic rather than

\* *Dublin Medical Press*, July 6th, 1859. Translation from the *Bibliothek für Lager*, by Dr. W. D. Moore.

† *Opus citat.*, p. 376.

strong; it is remarkably liable to exacerbations from emotional excitement. Trousseau has, in one instance, seen projection of the precordium from long continued palpitation. The sounds of the heart are usually sharp and clear, as in ordinary nervous palpitation. A soft systolic murmur, more or less persistent, but rarely of long continuance when not dependent upon valvular lesion, is frequently heard at the apex of the heart. A murmur of similar quality and rhythm still more frequently exists at the base, either in the aorta or in the pulmonary artery; but it is not transmitted into the arteries of the neck. The former of these murmurs is most probably adynamic, the latter hæmic; both are occasionally harsh in quality. I have frequently heard, in these cases, *sounds* both single and double in the carotid arteries, but never murmur, where it was not due to pressure from the enlarged thyroid gland. Trousseau has observed, as a constant symptom in exophthalmic goître, the "cerebral macula," viz., a reddening of the skin after slight irritation, as in cerebral or ataxic typhoid fever. Foot, likewise, noted this phenomenon in one of his cases.\* An elevation of temperature of 1° to 2° F., during the paroxysm, has been noticed by Basedow and Teissier, and also by Cheadle.† These symptoms have been attributed by Trousseau to debility of the vaso-motor nerves. Menstruation is usually irregular and defective, but the three prominent symptoms of the disease are, in most cases, exhibited before the occurrence of this derangement. The order in which, relatively, the symptoms were exhibited in my 6 cases‡ is shown in the following table:

TABLE XV.—EXOPHTHALMIC GOÎTRE.

Symptoms.	Order of Occurrence.				Total Cases
	First.	Second.	Third.	Fourth.	
Palpitation ...	4	0	1	...	5
Thyroid Enlargement ...	2	1	3	0	6
Projection of Eyes ...	0	4	1	1	6
Deranged Menstruation	0	1	1	4	6

So far as a conclusion may be drawn from these 6 cases, it

\* *Irish Hospital Gazette*, vol. ii., p. 163.

† *St. George's Hospital Reports*, vol. iv., p. 183, *Ibid.*, vol. vii., 1875, p. 81.

‡ I have, unfortunately, mislaid the notes of the 7th Case, referred to at p. 490.

would appear that palpitation is most frequently the primary symptom, and next, in order of frequency, enlargement of the thyroid gland; that projection of the eyeballs is the secondary symptom in the great majority of cases; that thyroid enlargement is, but in a somewhat less proportion, the usual tertiary symptom; whilst derangement of menstruation constitutes, in most cases, the fourth symptom in the order of occurrence. The number is, however, obviously too small to furnish a reliable basis for induction.

The enlargement of the thyroid gland constitutes the most urgent symptom of the disease. It is usually slow but fitful in progress, the right lobe being, in most cases, the first portion affected, and attaining larger dimensions than the remainder of the organ.\* Its primary increase is due to enlargement of the thyroid vessels; hence pulsation and *bruit de soufflet* during the period of vascular excitement, after which partial subsidence of the swelling is seen to occur. At a later period the parenchyma and the connective tissue of the gland are found to have undergone true hypertrophy; the organ is now more solid, and the increase of bulk has become permanent.

In some cases the excess of vascularity and the activity of pulsation of the thyroid body have been such as to lead to an error of diagnosis. Dr. Stokes has known the tumor to be mistaken for aneurism, a day having been actually appointed for the operation of tying the carotid artery before the error was detected.

Huskinness of voice, and even aphonia and stridor, are not unfrequently produced by the pressure of the enlarged thyroid upon the trachea and recurrent nerves.

Life has been actually endangered by mechanical pressure upon the trachea and blood vessels. Trousseau mentions the case of a boy in whom tracheotomy was contemplated as the only means of preventing suffocation; but relief was obtained by bleeding from the arm, the application of ice to the tumor, and the administration of digitalis.

I have, in one instance, seen the thyroid enlargement attain such

\* The right lobe was larger than the left in 3 of Moore's 6 cases, and likewise in the case recorded by Banks. *Dublin Hospital Gazette*, vol. ii., p. 129.



dimensions, as to place the patient's life in the utmost danger from asphyxia and coma by pressure on the trachea and the jugular veins. The patient, a female, aged eighteen, was admitted into hospital under the care of my late colleague, Dr. Curran, on the 10th of January, 1870, with all the symptoms of Graves' disease well pronounced. The thyroid gland had already attained the size of half an ordinary melon; it pulsated violently, and, by pressure upon the jugular veins and the trachea, caused great congestion of the neck and face, and urgent dyspnoea. The eyes projected to an extreme degree, and the patient was unable to swallow solids of any kind. Ordinary treatment, including bleeding from the arm, was attended with only temporary relief. The swelling increased, the patient became delirious, and asphyxia was imminent. The tumor being remarkably tense, it was suggested in consultation that relief might be obtained by division of the cervical fascia. This operation, which was attended with no ordinary difficulty owing to the great number and size of the superficial veins, was successfully performed by my colleague, Mr. Hayes, on the 27th of February; the fascia was slit up upon a director along the mesial line, from the *fourchette* of the sternum to the os hyoides; the gland protruded through the wound, divaricating the edges of the incision to an extent of several inches, and the patient experienced immediate relief. The loss of blood did not exceed eight ounces.

On the following day the cervical fascia was further divided, and the sterno-hyoid and sterno-thyroid muscles, which were on the stretch, were partially cut through. Relief was now complete; the patient was enabled to lie down and to take solid food.

She was discharged on the 12th of April, six weeks and three days after the date of operation. The catamenia, which had been irregular, were re-established. The thyroid enlargement became soft, gradually diminished in size, and the wound cicatrized. The patient visited the hospital in May, 1871, viz., a year subsequent to operation. She was then in fairly good health, the thyroid tumor being not larger than a small orange.\*

\* Vide *Medical Times and Gazette*, May 27th, 1871. I saw this woman again in April, 1875. The thyroid enlargement then scarcely amounted to disfigurement, proptosis no longer existed, menstruation was regular, she felt quite well and was actively employed, but the pulse was slightly accelerated.

The appearance of the eyes constitutes the most striking characteristic of the disease; they are prominent and staring, as if under the continuous strain of a great physical effort, and impart to the face a maniacal expression. The pupils are, with few exceptions, usually dilated, and the conjunctival and sclerotic veins are more or less turgid. This condition of the eyes is exaggerated during a paroxysm of palpitation. It is usually symmetrical, but in a case recorded by Desmarres, one eye only was affected. Vision is in many cases misty, and the focal distance is usually shortened; but frequently there is no alteration of focus or of visual perception. Trousseau mentions a case, recorded by Pain, in which one of the eyes was actually dislocated from the orbit by pressure from behind.

The prominence and tension of the eyeballs have been attributed to three different causes; namely, excess of the vitreous humour; hypertrophy of post-ocular connective tissue, with œdema, or accumulation of fat; and dilatation and distention of the post-ocular vessels. In one instance Trousseau found the globes, which were of ordinary size, thrust out of the orbits by excess of areolar and adipose tissue.\* In Basedow's and Keusinger's cases the eyeballs were atrophied, and the orbital fat was in excess.

As to the etiology of the disease, I consider Dr. Lauder Brunton's hypothesis the most tenable.† It is not only in consonance with the admitted condition of the carotid and thyroid arteries, but it explains the increase of ocular protrusion exhibited under vascular excitement, and the partial subsidence of the eyeballs which follows the cessation of a paroxysm. In a case recorded by Mr. Sharpley,‡ the retinal veins were seen to pulsate during a paroxysm.

After death, sanguineous extravasation has been found in the retina, the vessels of the choroid being congested. Pigmentary deposit in the retina has been detected in at least three instances; viz., in the case recorded by Withusen, in one examined by Dr. Argyll Robertson,§ and in a third case recently

\* *Opus citat.*, p. 582.

† *Vide* p. 1051.

‡ *Medical Times and Gazette*, September 5th, 1874.

§ Dr. J. Begbie, *Contributions to Practical Medicine*, p. 175 (foot note).

under the observation of Dr. Nixon in the Mater Misericordiæ Hospital.\*

Anæmia, and in the female, leucorrhœa, are pretty constant symptoms of exophthalmic goître. They are, however, by no means necessary, or even ordinary precursors of the "symptomatic triad," as suggested by Luton. In four of the cases published by Dr. Moore, anæmia did not exist; three of these patients were actually florid. Niemeyer does not admit a necessary connexion between anæmia and exophthalmic goître.† Respiration is easily quickened, and not unfrequently it is permanently accelerated. There is insomnia, and the bowels are usually constipated. In the advanced stages the appetite for solids is indifferent, or entirely lost, and in many cases persistent vomiting exists. Intercurrent diarrhœa, epistaxis, intestinal hæmorrhage, and œdema of the feet, are occasionally witnessed in the course of the disease.

Doctor Graves has remarked the frequent association of hysteria with this disease; he has even suggested the dependence of the hysterical globus upon temporary enlargement of the thyroid gland. The connexion is, however, doubtful.

The disease is rarely fatal. When death results from it, dilatation of the heart with its consequences, congestion of the lungs, anasarca, and effusion into the serous cavities, is usually the immediate cause; but not unfrequently the patients succumb to intercurrent affections of the respiratory organs, to chronic renal disease, or to cerebral hæmorrhage. Death has likewise resulted from erysipelas, and from gangrene following anasarca of the lower extremities.

The recorded *post mortem* examinations are few. Besides those given by Trousseau, Cruise and McDonnell, Basedow, and Keusinger, already referred to, examples have been published by Marsh,‡ Smith,§ Withusen,|| Praël,¶ Banks,\*\* Reith,††

\* This case has not been as yet published.

† *Opus citat.*, vol. i., p. 375.

‡ *Proceedings of the Pathological Society of Dublin*, 1841.

§ Stokes, *Diseases of the Heart and Aorta*, p. 291.

|| Trousseau, *opus citat.*, p. 570. ¶ *Ibid.*, p. 570.

\*\* *Dublin Hospital Gazette*, June 1st, 1855.

†† *Medical Times and Gazette*, November 11th, 1865.



J. Begbie,\* and Flint.† Other fatal cases are referred to by Cheadle‡ as having been recorded in Germany by Virchow and von Recklinghausen, and in France by Péter.

The case published by Dr. Banks claims a more extended notice, as being one of the most complete on record. The patient, a female, aged thirty, when fifteen years old, suffered from mental disquietude and emotional excitement. Her menstrual function had been always irregular, and for the last twenty months of her life it was entirely suspended, having been, about that time, suddenly arrested in the midst of a period. She had been subject to palpitation, and in the winter to bronchitis.

The eyes were prominent, brilliant, and staring; but they were not congested, nor was vision in any way affected.

The thyroid gland was much enlarged, the right lobe in a greater degree than the left; it increased in size during palpitation and fits of coughing, and it yielded a purring thrill, and a loud continuous venous hum. There was arterial throbbing in the neck, accompanied by a single and interrupted arterial sound.

The pulse was rapid, feeble, and unequal; it was likewise irregular and intermittent. Precordial dulness was extended, and the action of the heart was irregular and tremulous, but no cardiac murmur existed.

Towards the close of the patient's illness there was œdema of the lower extremities and eyelids; the urine contained albumen and some blood, and, in the recumbent posture, there was great respiratory distress whilst the cervical throbbing and murmur became nearly imperceptible. Death occurred by syncope. The *post mortem* appearances I shall record in the author's words:

"The thyroid gland, enlarged to four or five times its natural size, was found to cover, to a considerable extent, the front of the trachea. The right lobe was larger than the left; the thyroid veins were remarkably dilated; the gland was dense, very

\* *Dublin Hospital Gazette*, vol. ii., p. 109, from Case Book of *The Edinburgh Medical and Surgical Journal*.

† *Diseases of the Heart*, third edition, p. 311.

‡ *St. George's Hospital Reports*, vol. iv.



solid to the feel, and lobulated. A section of different parts disclosed the existence of numerous cysts, containing a yellow fluid like honey. The contents of some of the cysts were dark coloured, and resembled coagulated blood. The microscopic appearances were similar to those observed and figured by Rokitsansky in ordinary enlargement of the thyroid.

"The jugular veins were enlarged. The bronchial glands were found of an unusually large size. The heart was enlarged generally, and the cavities dilated, but not to any considerable extent.

"The heart was as large as that of an ordinary man, the subject being a woman under the middle size. The valves were free from disease, with the exception of slight thickening of the anterior edge of the mitral valve.

"The lungs were highly congested, and the bronchial membrane bore the marks of intense inflammation. The liver appeared as if in the earliest stage of cirrhosis; the spleen large and congested.

"The brain softer than natural; the lining membrane of the ventricles much thicker than usual. The kidneys had undergone the changes usually observed in the early stages of Bright's disease."

In Dr. Begbie's case, which was that of a man, aged thirty-one, in which all the symptoms of the disease existed, death occurred from congestion of the lungs and general serous effusion. The heart was dilated and thinned; it was distended with fluid blood. The jugular veins were dilated, that of the left side being an inch and a-half in diameter; the thyroid was enlarged; the spleen and kidneys were likewise enlarged; the liver was cirrhotic.

I am indebted to Dr. Cruise for the following report of the case, already referred to, which came under his notice: "Margaret M., aged thirty-six years, mother of several children, entered the Mater Misericordiæ Hospital under my care, September 26th, 1864. Her case was a well marked one of exophthalmic goître. The disease was of a few months' duration, and came on while wet nursing a child in Lord L.'s family. From the time of her admission, up to her death, November 30th, 1864, she steadily sank. Palpitation was most violent, and uncontrolled by the

remedies used (viz., digitalis, bromide of potassium, iodine, and cold to the thyroid, as advised by the late Dr. Aran of Paris). During the last few days of her life she passed into a semi-conscious state, with sordes on the teeth and gums, in fact a typhoid state, with remarkable dilatation of the pupils, which contracted markedly with the contraction which existed at first. On examination after death, the heart was found slightly hypertrophied, but otherwise sound. The thyroid was enlarged, but not to the extent observed during life, and the eyeballs were healthy. The only marked change discovered was in the sympathetic nerve in the neck, the ganglia of which, on both sides, were so atrophied as to be barely distinguishable from the shaft of the nerve. On examination under the microscope (made by Dr. Robert McDonnell) the ganglia were found to consist of little more than connective tissue and fat, none of the ganglionic structure being visible."

In Dr. Reith's case, the middle and inferior cervical ganglia of the sympathetic were enlarged, hardened, and infiltrated with granular matter; the nerve itself between these ganglia was likewise enlarged, and the surrounding cellular tissue was thickened. Dr. Reith was disposed to regard these changes as essentially strumous.

A question of great importance, as bearing upon the pathology of exophthalmic goitre, is the precise state of the heart in its advanced stages.\* Towards the settlement of this question morbid anatomy has contributed but very scant information. In all the recorded autopsies, one or both ventricles were found to be dilated or hypertrophied; but, in every instance, with the exception of those by Drs. Banks and Cruise just given, valvular or aortic disease, adequate to account for these conditions, likewise existed. Hence, clinical observation constitutes almost the only available evidence from which a conclusion can be drawn.

The apex of the heart has been displaced outwards in every chronic example of the disease. In most of the recorded cases the first sound of the heart was sharp and clear, whilst the radial pulse was feeble. In one of my cases (No. 132, p. 1068) reduplication of the second sound existed without pulmonary con-

\* This subject has been already discussed at p. 489.

gestion, whence I inferred dilatation of the left ventricle. From these data, the conclusion may, I think, be legitimately drawn, that the left ventricle had become in some degree dilated. I am, therefore, in agreement with Dr. Stokes on this question, and I think the evidence to the contrary adduced by Trousseau is far from conclusive. Such is, likewise, the opinion of Virchow and Niemeyer. I cannot concur in the opinion of M. Luton, that the right side of the heart is always passively dilated in this disease;\* nor even if such were the case, could I admit his conclusion that the symptoms which characterize the affection would be explained thereby.

The *diagnosis* of exophthalmic goître, in its typical form at least, cannot be attended with difficulty. It is liable to be confounded with ordinary bronchocele only; but from this it differs, as pointed out by Stokes, in not being connected with soil or climate, and in the remarkable variations of volume exhibited by the tumor. The enlargement of the thyroid, moreover, seldom attains to the magnitude exhibited in bronchocele; and, as remarked by Graves, it becomes stationary at a stage when the growth of bronchocele is accelerated. The swelling does, however, contrary to the opinion of Graves, not unfrequently "amount to actual deformity." The remarkable throbbing of the carotid arteries usually witnessed in this disease, may be confounded by a careless observer with that due to inadequacy of the aortic valves; but the absence of a diastolic murmur at the base of the heart, and the character of the radial pulse, will suffice to exclude the latter affection. The prognosis is, on the whole, not unfavourable, but it must be made with reference to the actual condition of the patient. Most of these cases improve and relapse alternately, the disease becoming chronic, and ultimately stationary. Death seldom results directly from it.

The *treatment* must vary, according to the view taken of the pathology of the disease, and the special indication afforded by symptoms.

The resemblance which, in one feature, it bears to bronchocele, has suggested the use of iodine, and several examples of cure by

\* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques*, tom. xvi<sup>e</sup>, p. 506.



means of this agent have been recorded.\* Trousseau, however, believes that iodine is positively injurious. Iron may be given with benefit where anæmia predominates, if the pulse-rate be not excessive.† In the latter case, especially if the pulse be at the same time weak and intermittent, the impulse of the heart feeble, and the first sound clear but faint, digitalis is the appropriate agent to be employed. Under its use, in doses of ℞ to ℞xv of the tincture given thrice daily, with the spirit, or compound tincture, of chloroform, the pulse-rate is rapidly reduced, whilst the action of the heart acquires strength and steadiness. Digitaline may be substituted, in doses of one-fortieth of a grain every sixth hour till the above mentioned effects are exhibited, or, should the stomach prove intolerant of the drug in any form, ʒj of the *unguentum digitalis* of the *Hamburgh Pharmacopœia* may be smeared over the precordium night and morning, the result being carefully observed. In every case the use of the drug should be suspended when the pulse has been reduced below 100 in the minute. Trousseau, however, thinks digitalis should be given till the pulse-rate falls to 70, and the earliest symptoms of its toxic effects are exhibited; viz., vertigo and nausea. This is unnecessary, and not free from danger. It is, moreover, objectionable, as precluding the repeated use of the medicine, without which permanent benefit cannot be obtained.

Aconite may be given with advantage during the paroxysms of palpitation, if the impulse of the heart be vigorous and the pulse free from intermission. Bromide of potassium may be likewise administered with benefit in such cases.‡ Gelseminum, arsenic, and iodo-bromide of calcium§ have been used in the treatment of exophthalmic goitre; galvanization of the cervical sympathetic, as first resorted to by Dusch, has been likewise tried, and with satisfactory results, by Chvostek, Moritz Meyer, and by Eulenburg and Guttmann.||

One of the primary objects of general treatment in cases at-

\* *Vide Stokes, opus citat., and Cheadle, St. George's Hospital Reports, vols. iv. and vii., also Case 134, p. 1071.*

† *Vide Foot, Irish Hospital Gazette, vol. ii., p. 165, and Sharpley, loco citat.*

‡ *Vide Case 130, p. 1066.*

§ *Dr. Guptil, American Journal of the Medical Sciences, January, 1874.*

|| *Die Pathologie des Sympatheticus, 1873.*



tended with derangement of menstruation, should be to re-establish this function. Improvement generally coincides with the return of the menstrual flux at the monthly periods, and, as remarked by Charcot,\* such is likewise the case on the occurrence of pregnancy.

Cold water bathing, and especially cold showers, at the proper season, and during the intervals between the monthly changes, are of great value as a means of allaying nervous excitement and promoting general nutrition.

Bleeding from the arm has been followed by complete relief during a paroxysm of dyspnœa in which strangulation from vascular engorgement of the thyroid body had been imminent. Ice should be applied over the tumor, at the same time, as suggested by Aran and Trousseau.†

In extreme cases, tracheotomy has been suggested as the only means of saving life; but, irrespectively of the unfavourable prospect of ultimate recovery which this operation affords, even though successful in rescuing the patient from immediate death, it is attended with the utmost difficulty and danger in the performance, owing to the enlargement and great vascularity of the thyroid body. I have, in a previous page (1057) indicated an alternative procedure, which was eminently successful in the only instance in which it has been resorted to; namely, division of the cervical fascia over the tumid gland.

The following six cases constitute the only examples of exophthalmic goître of which I have preserved full notes.

CASE CXXIX.—*Exophthalmic Goître and Impairment of Vision; Palpitation, with Murmur in the Pulmonary Artery; Rapid Pulse; Irregular Menstruation.*

Anne C., aged thirty years, visited the Mater Misericordiæ Hospital as an extern patient, May 31st, 1867. Three years previously she had been in hospital for pain in the left shoulder. About the same time she noticed a swelling in her neck, and her sight began to fail. During the last year menstruation had been

\* *Gazette Hebdomadaire*, 1862.

† *Bull. de l'Acad. de Médecine*, December 4th, 1860, tom. xxvi., p. 122.

irregular, and on two occasions it was suspended for two months. The eyes were prominent, and vision was imperfect in the left; it was "misty" and "cloudy" after exertion. There was general enlargement, with pulsation of the thyroid gland. The carotid arteries pulsated visibly; the pulse was 168 and weak, and the sounds of the heart were sharp, clear, and free from murmur. She was directed to take, twice daily, gr. ss of extract of belladonna, with an equal quantity of extract of nux vomica.

June 15th, 1868. She again presented herself in the out-patients' room, and the following note was taken. Pulse small, 160; heart's action "hammering," and in rate corresponding to the radial pulse, but not attended with murmur. Thyroid enlarged and pulsating; eyes prominent, and sight weak; respiration 36; has cough, and is remarkably nervous.

October 7th, 1868. The report made was as follows. Pulse 168; *bruit de râpe* over pulmonary artery, but not audible elsewhere. Menstruation has been suspended for the last two months; is deaf in right ear. To have bromide of potassium, grs. x, thrice daily.

January 13th, 1869. The last note made of this case bears the above date, and is to the following effect. Pulse 150; eyes prominent; thyroid large and pulsating; heart bounding.

It is noteworthy that impairment of vision not only existed in one eye, but was coeval with protrusion of the eyeballs, and was one of the earliest symptoms observed.

CASE CXXX.—*Exophthalmic Goître; Palpitation, with Roughness of the First Sound, and Throbbing of the Carotids; Irregular Menstruation; Improvement.*

Eliza E., aged twenty-two years, a domestic servant, of fair complexion, and a native of Birmingham, visited the extern department of the Mater Misericordiæ Hospital on the 10th, and again on the 17th of January, 1868. She had been for several years suffering from palpitation, which first occurred after severe mental affliction. Six months previous to her visit she noticed that her eyes projected, and three months later, that her neck

was swollen. Pulse 132; eyes remarkably prominent and staring especially the left, which was myopic. The thyroid body was enlarged, and pulsated strongly. There was palpitation of the heart, with roughness of the first sound, and visible pulsation of the carotids accompanied by a systolic arterial murmur. Menstruation was irregular, the periods varying from four to six weeks. Appetite, sleep, and state of bowels natural; tongue clean. To have grs. vij of bromide of potassium thrice daily. A few months later this girl reported herself as greatly improved, and able to resume her employment.

Palpitation preceded the other two events in this case, and protrusion of the eyes was second in order.

CASE CXXXI.—*Palpitation; Exophthalmia; Enlargement and Great Congestion of Thyroid Body; Systolic Murmur in Thyroid Gland and in Carotid Arteries; Dyspnœa; Dysphagia; Cough and Hæmoptysis; Œdema of Feet, and Occasional Syncope; Rapid Pulse; Intermittence of the Heart; Masking of the Second Sound; Extension of Precordial Dulness, and Displacement of Apex Outwards.*

Margaret M., aged forty years, a dressmaker, married, and has had nine children, is still "regular," visited hospital, July 6th, 1869. Her illness commenced with palpitation eight years previously, and about the same time her eyes became somewhat prominent. Four years later her breathing became short, and about the same time she began to experience some difficulty in swallowing solids. For the last two years she has been suffering from cough with mucus expectoration, which, on three several occasions, was mixed with blood. Epistaxis has occurred occasionally. During the heat of the present summer she had been prevented, by dyspnœa, from assuming the recumbent posture, and for the last fortnight she has been drowsy.

When first seen by me her condition was the following: The eyes were remarkably and equally prominent, conjunctivæ congested, and pupils contracted. She possessed the power of closing the eyes by an effort, but during sleep they remained open. Both lobes of the thyroid body were greatly enlarged, and there

was congestion of the veins of the neck, which was much increased when the patient coughed. Indeed, she declares that during the act of coughing, the tension is so great over the thyroid gland, that she feels as if it must "burst." The root of the neck was tumid and rounded off. She did not suffer from headache. Bowels confined. Respiration was shallow, and 60 in the minute. The pulse was 108, and weak. The action of the heart was characterized by occasional intermissions; the sounds were free from murmur, but the second was not clear at any point of the precordium. The respiratory sounds were weak, but equal on both sides. Percussion-resonance normal. To have aperients, and grs. x of bromide of potassium, thrice daily.

July 30th. She has had slight weakness, with temporary loss of vision and of consciousness, two or three times daily for the last fortnight; this was not caused by exertion.

April 6th, 1870. She has been "irregular" for the last six months, and the feet have been swollen at night for the last four months. Pulse 120. Eyes prominent and lachrymose, and when she coughs forcibly she feels them tense, and as if she could not close them.

The thyroid body was much enlarged, and crossed by turgid veins; it yielded a loud systolic bellows-murmur. A murmur of similar character and rhythm was audible in the carotid arteries. The face was congested. Precordial dulness was extended; apex-pulsation in nipple-line, and impulse strong.

This patient ceased to attend at the hospital, and I lost sight of her.

CASE CXXXII.—*Palpitation and Rapid Pulse; Exophthalmia; Enlargement and Pulsation of Thyroid; Throbbing of Carotids; Derangement of Menstruation; Abrupt Cardiac Impulse; Sounds Clear, and Free from Murmur; Second Sound Reduplicated at Midsternum; Dilatation of Left Ventricle.*

Miss McC., a farmer's daughter, aged twenty-three years, consulted me on the 13th of May, 1870. Had undergone much labour in milking cows at a large dairy. Two years previously she began to experience palpitation; about the same time she



noticed swelling at the root of her neck, and enlargement of her eyes, both of which changes have been gradually increasing since that date. Has had, for the preceding six months, slight tenderness of the left knee, at a point some distance above the joint.

When she came under my notice, her skin was dusky and her eyeballs were prominent, giving her a remarkable staring expression, which her brother, who accompanied her, characterized as a "queer look." The thyroid body was enlarged and pulsatile, but, as I was informed, it varied inversely as the activity of the menstrual function. There had been no "change" for the preceding two months, and the thyroid was, at the time of examination, remarkably large. Pulse 144, and regular; throbbing of the carotid and radial arteries; the impulse of the heart was abrupt and "hammering"; the sounds were clear and unattended with murmur, the second being reduplicated at midsternum.

To have bromide of potassium (grs. x) thrice daily, and compound aloetic pill (grs. v) every night. To give up the use of strong tea, to which she had been addicted. I have had no further report of this patient. In the absence of the signs of pulmonary congestion in this case, I regarded reduplication of the second sound as evidence of dilatation of the left ventricle. The clear and sharp character of the sounds tended to confirm this view.

CASE CXXXIII.—*Palpitation; Exophthalmia, and Enlargement of Thyroid Body, with Pulsation, Thrill, and Systolic Bruit; Bruit de Diable; Weak and Rapid Pulse; Tumultuous Action of Heart, with Systolic Murmur both at Apex and Base; Amenorrhœa; Great Dilatation of the Pupils; Emaciation; Temporary Improvement, and Relapse.*

Doctor Stokes was kind enough to invite me to see, with him, a remarkable example of this disease, on the 28th of April, 1871. The patient was a young lady from the country, aged twenty-four years. She gave the following history of her illness: Two years previously she had been much frightened, and immediately afterwards she began to suffer from palpitation. About a week subsequently her eyes began to project, and the root of her neck

to swell. Her menstrual function now became deranged ; she became nervous and sleepless, and began to lose flesh.

Under a course of treatment with tonics and chalybeates she improved considerably ; the swelling of the thyroid subsided, the eyeballs receded, the palpitation was mitigated, and menstruation was re-established.

This improvement was of brief duration, and when I saw the patient her condition was as follows : She was greatly emaciated, pale, and nervous ; the eyeballs projected quite beyond the lids ; she closed the eyes with great difficulty, the effort to do so being attended with circumorbital pain, and she slept with the eyes open. The pupils were greatly dilated, and when the patient sat with one side of her face to the window, light entering by the pupil and reflected from the *fundus oculi*, was distinctly transmitted at an angle through the sclerotic.

The thyroid body was much enlarged, especially on the right side, and coursing over it were seen large and distended veins which pulsated synchronously with ventricular systole ; this pulsation was arrested by light finger-pressure at the root of the neck.

A loud intermittent *bruit*, of a buzzing character, and synchronous with ventricular systole, was audible over the thyroid body, and at its posterior margin a combination of triple sounds, consisting of carotid *bruit de choc*, jugular *bruit de diable*, and the thyroid buzz just described, was heard. The thyroid body pulsed sensibly, and communicated to the hand a faint thrill. Pulse rapid and weak ; cardiac impulse strong and heaving, and both at apex and base a faint but rough systolic murmur existed. The apex-pulsation was in the nipple-line. She had not menstruated for the last four months. The bowels were regular.

Small doses of citrate of iron and quinia were prescribed, and inunction of the precordium with the ointment of digitalis (Hamburgh Pharmacopœia) was directed. I had not again an opportunity of seeing this lady, and I have heard nothing further of her case.

CASE CXXXIV.—*Menorrhagia ; Palpitation and Rapid Pulse ; Great Enlargement of Thyroid Body, and Extension of Swelling into Anterior Mediastinum ; Enlargement and Distention of Cervical, Thoracic, and Brachial Veins ; Throbbing of Carotid Arteries ; Systolic Bruit de Soufflet in Thyroid ; no Exophthalmia, but Temporary Obscuration of Vision ; Improvement under Treatment with Iodine.*

Miss O'B., aged about forty years, consulted me in September, 1872, She had menstruated regularly, but with pain. Her neck began to swell six years previously, but from this she felt no inconvenience till nine months prior to her visit to me. When first noticed, the swelling of the neck was chiefly on the right side.

In January, 1872, she was suddenly attacked with "flooding," which lasted continuously for twenty-four days, the discharge, consisting partly of liquid, and in part, of clotted blood. The loss was very considerable, and greatly reduced her strength. After this occurrence she began to suffer from palpitation and dyspnoea on slight exertion, and the swelling in the neck rapidly increased.

At the date of this lady's visit to me the thyroid body had attained a large size, and, as judged by the percussion-dulness and the prominence of the sternum, a tumor continuous with it extended fully two inches into the anterior mediastinum.\*

The superficial veins at the root of the neck were greatly enlarged and tortuous, the external jugular being quite as large as the internal jugular in its normal state, and from the thyroid cartilage to the *fourchette* of the sternum a vein of equal size coursed vertically along the middle line. Over the thyroid body generally a loud systolic *bruit de soufflet* was audible. The cervical arteries pulsated visibly, and the superficial veins of the arms and of the upper portion of the chest on both sides were enlarged and distended. The upper portion of the sternal region, for about two inches downwards, was prominent and dull on percussion as already stated, whilst both subclavicular regions were

\* A case, which ultimately proved fatal, exhibiting a similar extension of the thyroid into the chest, is reported from Guy's Hospital, in the *Medical Press and Circular* of March 23rd, 1870. The heart was normal in this case.

depressed, and, in the latter situations respiration was feeble. Over the sternal prominence a loud musical stridor was heard with both inspiration and expiration.

The heart pulsated in the normal position; its action was rapid and tumultuous, but the impulse was rather abrupt than strong, whilst the second sound was sharp, clear, and free from murmur.

The pulse was regular but abrupt, and in the sitting posture it averaged 144. The eyes were not prominent, but on the occasion of her third visit, viz., on the 1st of October, she experienced a mist passing over her eyes from below, when she looked upwards. There was then slight cedema of the feet and of the right arm and shoulder.

The slightest exertion caused much difficulty of breathing. Sleep was disturbed, and she experienced some discomfort at the root of the neck when swallowing solids. Voice husky, and singing voice entirely lost. Face of a leaden hue. To have grs. v of iodide of potassium, with ℥ij of tinct. of iodine, thrice daily, and the tumor to be painted with the liniment of iodine. To have warm sea-baths, and subsequently open sea-bathing. Digitalis had been given in large doses, with very little benefit.

I saw this lady again in the following year. She had continued the iodine treatment during the interim. The thyroid swelling was now much reduced, and the venous engorgement less. She had not had a return of the flooding; her monthly health was now regular, and her strength had improved. I have not since heard of her.



## CHAPTER X.

### DISEASES OF THE AORTA.

THESE diseases may be classified under the three heads of

- (a) Atheromatous change, in its several stages.
- (b) Simple dilatation.
- (c) Aneurism.

*Atheromatous transformation* of the coats of arteries is a result of malnutrition, and essentially degenerative. Not unfrequently the products of a low form of inflammation of the internal and middle coats are combined with those of tissue degeneration. Moxon says, "Atheroma is in continuity with arteritis, and graduates from a condition in which no inflammatory results can be found, into one in which inflammation is unmistakably present." He holds that inflammatory irritation of arteries arises from vascular tension, and remarks that diseases which lessen the volume of the blood, and therefore diminish the tension of the arteries, such as phthisis and mitral obstruction, are very rarely associated with atheroma.\* Holmes considers strain of the walls of arteries to be the principal factor in the production of atheroma.†

Niemeyer maintains that antecedent chronic inflammation is a necessary condition of atheromatous change.‡ Virchow is of the same opinion; adding, that the primary irritation results in enlargement of the connective-tissue corpuscles, multiplication of nuclei, cell-proliferation by endogenous growth, and, finally, fatty degeneration of these new products.§

The degenerative changes in the coats of blood-vessels constituting atheroma, consist essentially in fatty and calcareous meta-

\* *Guy's Hospital Reports*, vol. xvi., third series, 1871, p. 431.

† *System of Surgery*.

‡ *Text-Book of Practical Medicine*, vol. i., p. 345.

§ *Cellular Pathology*, translated by Chance, p. 361.

morphoses, as shown by Gulliver,\* and admitted by Paget,† Erichsen,‡ and all modern pathologists. Aitken regards atheroma as a fatty product of degeneration of tissue; but he considers rheumatism, gout, and syphilis, to be the ordinary causes of this change.§ Hodgson has noticed the frequent occurrence of aneurism in persons who had suffered from syphilis and taken large quantities of mercury.|| The doctrine of syphilitic causation of atheroma is, however, very questionable. It is noteworthy, that whilst syphilis is more common in civic than in rural populations, aneurism is not proportionately in excess amongst the former; and, as remarked by Mr. Cooper Todd, whilst syphilitic affections were formerly more common and more virulent amongst soldiers than at present, aneurism has been latterly rather on the increase in the army.¶

The influence of gout in the production of atheroma cannot, I think, be doubted. The degenerative changes, which, in chronic gout, are exhibited by the several structures in and around the joints, are kindred to, and most frequently associated with, corresponding changes in the walls of the arteries. I cannot recognise rheumatism as a cause of atheroma, except in so far as it may be associated with gout, under the form of "chronic rheumatic arthritis."\*\*\*

In many cases atheroma is directly and exclusively the result of persistent arterial tension from hypertrophy of the left ventricle.†† Hence, its frequent association with the cirrhotic form of Bright's disease, and with patency of the aortic valves. Hence, likewise, it is first exhibited in the arch of the aorta, which is most exposed to pressure from an hypertrophied ventricle. The change commences in the middle or fibrous coat, in the form of minute fawn-coloured spots; these gradually extend and coa-

\* *Medico-Chirurgical Transactions*, vol. xxvi., p. 86.

† *Lectures on Surgical Pathology*, vol. i., p. 138.

‡ *Science and Art of Surgery*, 1869, vol. ii., p. 1.

§ *The Science and Practice of Medicine*, fifth edition.

|| *A Treatise on the Diseases of Arteries and Veins*, 1815.

¶ *Army Medical Reports*, 1868.

\*\*\* *Vide A Treatise on Chronic Rheumatic Arthritis*, by Robert Adams, M.D., 1857.

†† *Vide* p. 513, *et sequent.*, and p. 824.

lesce, assume a deeper yellow tint, and become more dense and brittle. In the same proportion, the coats of the arteries lose their properties of resistance and elasticity, and the vessel is slowly and permanently dilated. The middle coat thus deteriorated may be rent by the pressure of the blood, or it may undergo the further change of calcification, forming scales, the sharp edges of which, as observed by Richet,\* may wound the "intima," and thus give rise to aneurism. The internal coat, when unsupported on the outside, from yielding or disintegration of the "media," is, in the great majority of cases, quickly broken through, and aneurism is the necessary result. The process of change above sketched, may, however, not extend beyond the stage of dilatation of the vessel. In the case of the superficial and smaller arteries, such as the radial and temporal, owing to their loose connexions and the tenuity of their walls, this is always accompanied by elongation and inflection.

Dilatation of the aorta, carried to a certain point, and involving the orifice of the vessel, if unattended with proportionate expansion of the valves, will give rise to patency and a murmur of reflux,† which, if the degree of inadequacy be extreme, will be prediastolic in rhythm.‡ If the internal surface of the vessel be rough from atheromatous change, a systolic murmur, located above the base of the heart, and not transmitted into the arteries of the neck, will be the result.§ I cannot admit that equable and moderate dilatation of the aorta is cognizable from physical evidence in the absence of the conditions and attendant signs just specified.

Dilatation of an artery carried to an extreme degree, and unaccompanied by breach of continuity in one or more of its coats, constitutes true aneurism; a form of the disease denied by Scarpa,|| but admitted by Corvisart,¶ Bertin,\*\* Laennec,†† and

\* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques*, 1865, vol. ii.

† *Vide* Case 87, p. 866.

‡ *Vide* p. 843, and Case 98.

§ *Vide* p. 839, and Case 62.

|| *A Treatise on Aneurism*, Wishart's translation, p. 54.

¶ *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels*, Hebb's translation, p. 302.

\*\* *Traité des Maladies du Cœur*, p. 93, *Observations* 37 and 38.

†† *Traité d'Auscultation Médiate*, tom. ii., p. 688.

by most modern authors. True aneurism must be recognized, but only as representing a state of transition; the size attained and the figure presented by it depending upon the degree and extent of antecedent disease in the walls of the vessel, and the strain exercised upon them. The distinction between dilatation and true aneurism, as recognized by modern writers, has reference rather to the figure than the degree of enlargement; a fusiform expansion of the vessel over a limited portion of its length, or a yielding of a portion of its circumference in the form of a sacular protrusion of all its coats, constituting true aneurism, whilst an equable enlargement, formerly designated cylindroidal aneurism, is now, according to its degree, recognized as dilatation of the vessel.

Aneurism, therefore, as I understand it, is either *true*, by which I would imply a localized bulging or protrusion of all the coats of the artery; *false*, one or more of the tunics being broken through; *compound*, where a false is engrafted upon a true aneurism by outgrowth from a limited portion of its surface;\* *dissecting*, constituted by rupture of the internal and middle coats, the blood having formed a new channel by separation of the outer from the middle coat over a limited portion of the circumference of the artery, and re-entered the vessel by a second opening, more or less distant from the original rent; and *varicose*, consisting in a communication between an aneurism and a vein, the pulmonary artery, or one of the chambers of the heart.†

False aneurism, the form in which the disease is most frequently presented, is usually the result of laceration or fissuring of the internal and middle coats of the artery, already deteriorated by atheromatous change. The blood insinuates itself through the rent so formed, and by its pressure determines the development of aneurism. Occasionally, as observed by Corvisart,‡ aneurism results from perforation of the inner coats of the

\* *Vide* Corvisart, *opus citat.*, p. 302, where an example is given.

† I do not propose discussing aneurism in its surgical aspects, and therefore I have not included in the above category, traumatic aneurism, aneurismal varix, or aneurism by anastomosis.

‡ Hebb's translation, p. 276.



vessel, by the rupture of an abscess situate between the middle and external coats. Laennec also admits this mode of origin of aneurism.\* False aneurism may be circumscribed or diffused. Circumscribed false aneurism has been divided into two species; namely, "mixed internal," and "mixed external." The "intima" protruding through a rent in the middle and external coats, and with the connective tissue forming the sac of the aneurism, constitutes the former species, the identity of which rests upon the authority of Dubois and Dupuytren based upon a single observation, and is now not admitted. The "mixed external," or the "aneurism of transition" of Cruveillier, is produced by rupture of the internal and middle coats of a previously diseased or dilated artery, the external coat, supported by the connective tissue, alone forming the wall of the sac. This is the most common form of aneurism, and that under which, except where other forms are expressly specified, the disease will be discussed in the succeeding pages.†

Diffuse false aneurism consists in an accumulation of blood outside the artery by rupture of all its coats, and limited only by the surrounding parts. It arises either from wound or laceration of the vessel by mechanical violence, or from rupture of the sac of a mixed external aneurism.

Aneurism is essentially, though not exclusively, a disease of middle age. Out of 120 cases reported by Lisfranc, 59 occurred between the ages of thirty and forty-five years;‡ and of 551 cases collected by Dr. Crisp, 398 were represented as having occurred between the ages of thirty and fifty years.§

Of 15 cases of aneurism of the aorta which have been under my observation and treatment, 11 were between the ages of thirty and fifty years, 3 were over fifty, and one only was under thirty years. Tables XVI. and XVII. comprise 84 cases of aneurism of the aorta; of these, 55 were between the ages of

\* *Traité d'Auscultation Médiate*, tom. ii., p. 688.

† Dr. Crisp (*Diseases of the Blood Vessels*, p. 108) mentions a variety of false aneurism formed by projection of the internal and middle coats through a rent in the outer tunic, and refers to an example of this kind in the Museum of the London College of Surgeons (No. 1642).

‡ Reichet, *opus citat.*

§ *Opus citat.*, p. 113.

thirty and fifty years, 10 were over fifty, and 4 were under thirty years; in 15 instances the age is not stated. The principal cause of the prevalence of aneurism at middle age must be the vigour and activity of the body at this period of life. Atheromatous change and calcification of the arteries is most common after the age of sixty, as stated by Bichat.\* But the smaller percentage, and the less degree of arterial change at an earlier period of life, are more than counterbalanced by the vascular strain arising from greater muscular power and habitual physical effort in middle age.

Sex has a manifest influence in the production of aneurism. Of the 551 cases referred to by Crisp, less than one-eighth had occurred amongst females, whilst my tables show only 10 females out of 84, or a little over one-eighth. The more laborious habits, and the greater muscular vigour of the male as compared with the female sex, by which vascular tension and strain are produced, may afford a satisfactory explanation of this difference. Mr. Porter has denied the influence of labour and muscular effort in the production of aneurism;† but, notwithstanding his great eminence as an authority on this subject, I cannot concur in the opinion just stated. Occupation, when ascertained in regard to a sufficient number of cases, may be accepted as a criterion by which to form a judgment on this question. My conclusions shall be drawn from examples of aneurism of the aorta exclusively; but manifestly they must apply equally to aneurism of the minor arteries.

Of 57 cases in Tables XVI. and XVII. in which the occupation of the patients is given, 12 were mechanics, 10 were soldiers or pensioners, 8 were labourers, 5 porters, 4 cabmen, 4 gentlemen, 4 housekeepers (females), 2 grooms, 2 policemen, 1 was a butler, 1 a sailor, 1 a dairyman, 1 a cattle drover, 1 a coal heaver, and 1 a strolling player upon a wind instrument. Thus, it would appear from the statistics at my disposal, that mechanics, soldiers, labourers, and porters, men whose avocations demand frequent and extreme muscular effort, or prolonged exercise under unfavourable conditions, enjoy an unenviable dis-

\* *Anatomie Générale*, tom. ii.

† *Observations on the Surgical Pathology and Treatment of Aneurism*.

tion in regard to liability to aneurism of the aorta. Indeed, in most instances, the first symptoms of aneurism can be traced to a definite occurrence, involving great strain or effort, or to a severe shock.

The relative frequency with which different arteries are affected with aneurism, would appear to depend upon (*a*) their magnitude and proximity to the heart, and (*b*) their liability to strain or injury, whether by the force of the blood current, or from mechanical causes. Hence the disease is most common in the aorta. It has occurred with about equal frequency in the innominate, carotid, subclavian, iliac, femoral, and popliteal arteries.\*

The greater liability of the aorta, as compared with other arteries, arises from its immediate exposure to the force of impulsion from the left ventricle. Owing to this circumstance, it is not only more liable to undergo those structural changes of its walls which predispose to aneurism, but such changes having been effected, it is more likely to suffer disintegration of structure from the force of the blood current. The figures representing the relative liability to aneurism of the different portions of the aorta, as deduced from the 84 cases comprised in Tables XVI. and XVII., are as follows : Ascending portion of arch, 30 cases ; abdominal aorta, 16 ; transverse portion of arch, 14 ; thoracic aorta, 8 ; descending portion of arch, 3. Thus, the frequency of aneurism of the ascending portion of the arch of the aorta was not only in excess of that in any other portion of the vessel, but equal to that of its occurrence in any *two* other portions of it. Two or more portions of the vessel were affected in the following relative frequency : Ascending and transverse portions of arch, 8 ; transverse and descending portions of arch, 3 ; ascending portion of arch and thoracic aorta, 2 ; transverse and descending portions of arch and thoracic aorta, 1 ; entire arch, 1 ; ascending portion of arch and abdominal aorta, 1.

As to the pathology of aneurism, I will only remark here, that I believe structural alteration of the coats, in a greater or less degree, always precedes simple or aneurismal dilatation of an artery ; and that vascular strain or tension is the immediate or

\* *Vide* Hodgson, *op. cit.* ; Lawrence, Lectures in *Lancet* and *Medical Gazette* ; Chomel, *Dictionnaire de Médecine* ; and Wardrop, *Cyclopædia of Practical Surgery*.

determining cause of its enlargement.\* Habitual vascular tension leads to impairment of nutrition, degenerative change, and loss of elasticity in the middle coat of the artery. Such changes being established, the rate of expansion of the disorganized portion of the vessel depends upon the degree of internal pressure to which it is subjected, and the frequency of repetition of such pressure. A high degree of blood-pressure suddenly brought to bear upon an artery in an early stage of structural alteration, may, within the period of a single act of respiration, produce the same result as a less degree of tension, exercised at intervals for many months, upon a vessel in a more advanced state of disease.

Where aneurism is the result of momentary strain, great muscular effort, or violent succussion of the chest or back, it is always of the false variety, and the patient is sensible of the occurrence of an accident, as of "something giving way" within the chest or abdomen. Sharp pain is felt at the seat of injury, the breathing is usually quickened, hæmoptysis not unfrequently occurs, and a sense of faintness, sometimes amounting to actual syncope, is experienced. In many instances in which the subsequent history of the case has proved the occurrence of rupture of some of the coats of the vessel at the moment of the imputed accident, the injured person has quickly recovered from the shock, and experienced but little inconvenience for months afterwards.† In all such cases, however, an appreciable deterioration of health, consisting in one or more of the following symptoms, will be found to coincide with the date of the accident; viz., shortness of breath on exertion, diminished power of endurance, fixed pain in the chest, back, or abdomen, or dry cough. In a few instances, after a period of incapacity for labour or exertion, but not characterized by definite symptoms, the injured persons have recovered ordinary health, and enjoyed complete exemption from suffering for long periods, after which symptoms of a definite character have been exhibited, and have continued with increasing severity, till the fatal termination of the disease.

The preceding observations may serve to explain the special

\* *Vide* Cases 135, 137, 138.

† Case 135, *c. g.*



liability to aneurism of the aorta, which is notoriously connected with certain pursuits and handicrafts requiring great and continued muscular effort with distended lungs, or fatiguing exercise with a constricted chest. Porters, sledgers, soldiers, and bandsmen, exhibit a much larger percentage of aortic aneurism than others not similarly employed. Aneurism of the aorta is usually fatal; but the examples of temporary arrest or permanent cure of the disease, recorded within the last ten years, are sufficiently numerous to encourage the hope of still greater success in its treatment. These remarkable results are partly due to the more accurate knowledge now possessed by physicians of the conditions which favour, and those which retard or prevent coagulation of the contents of the sac, and likewise, in some degree, to the discovery of new and more potent agents for effecting that object; but they are mainly to be accredited to the greater boldness and skill with which the resources of surgery have been latterly employed in the treatment of aneurism.

Owing to the indefinite character of the symptoms which, in many cases, attend aneurism of the aorta in its early stages, and still more to its frequent latency, especially within the thorax, it is difficult to determine the average duration of the disease in a large number of cases. Where, however, as has repeatedly happened, symptoms indicative of injury to the aorta are associated with a definite strain or concussion of the chest or back, and followed by those of aneurism of that vessel, the date of commencement of the disease may be fixed with accuracy. In the cases recorded in Tables XVI. and XVII., I find the duration of aneurism of the aorta within the chest to have varied from ten days to ten years; and that of aneurism of the abdominal aorta to have ranged from fifteen days to eleven years.

The longest duration of aneurism of the aorta witnessed by Walshe was thirty-eight months. Dr. C. J. B. Williams has mentioned the case of a gentleman then living, who, to his knowledge, had been the subject of aneurism for thirty years.\*

In general terms, the duration of aneurism of the aorta may be said to vary from a few weeks to as many years. It depends upon many circumstances, having reference to the constitution

\* *British Medical Journal*, May 12th, 1870.

and the contents of the sac, the size, situation, and bearings of the tumor, the complications of the disease, and the previous health and present habits of the patient. A true aneurism of small size, so situate as not to interfere materially with important organs, in a person of previously unimpaired health, favourably circumstanced in regard to treatment, and submissive to restriction, offers the most favourable conditions under which the disease can be presented. A false aneurism, lined to a great depth by solid laminated fibrin, and circumstanced as above mentioned, affords a scarcely less favourable prospect. Conditions the reverse of the foregoing, and proportionately to their respective gravity, would suggest an unfavourable prognosis.

As to the effect of aneurism of the aorta upon the left ventricle of the heart, opposite opinions have been expressed by pathologists; some eminent writers having held that it necessarily leads to hypertrophy,\* a view which, having reference to the impaired elasticity of the portion of the arterial wall constituting the sac, would seem theoretically to be well founded. Nevertheless, I am convinced that it is erroneous.

I believe that left ventricular hypertrophy exists in connexion with aneurism of the aorta, only where valvular disease or some other fully recognized cause of hypertrophy has been in operation.† In reference to this question, Tables XVI. and XVII. include 48 cases in which the condition of the valves, of the aorta, and of the left ventricle, is positively stated. Of this number, 8 exhibited disease of the aortic valves alone, or of the aortic and mitral valves conjointly; hypertrophy of the left ventricle existed in all these cases in connexion with aneurism of the aorta. Hypertrophy of the left ventricle, not associated with valvular disease, existed in 9 cases; but in 7 of these, extensive and advanced atheroma of the aorta coexisted with aneurism, and in 3 of the cases the hypertrophy was very slight. Atheroma of the aorta beyond the seat of aneurism was present in 20 cases in which hypertrophy of the left ventricle did not exist, and in 4 of these the left ventricle was in a state of atrophy, whilst, in

\* Hope, *Diseases of the Heart and Great Vessels*, third edition, pp. 435 and 450, foot note.

† *Vide*, p. 488.

12 others in which hypertrophy did not exist, the aorta was apparently healthy, except at the seat of the aneurism. In 2 instances the left ventricle was dilated and fatty (not hypertrophied) in connexion with extensive atheroma of the aorta. Thus, excluding the cases in which left ventricular hypertrophy was accounted for by lesion of the valves, 9 cases only, or a fraction over one-fifth of the remainder, can be adduced in support of the alleged dependence of hypertrophy of the left ventricle upon aneurism of the aorta. But, in most of these cases, the patients were mechanics or soldiers, and therefore exposed, in their daily avocations, to causes adequate to the production of functional hypertrophy; and in 7 of them the aorta was extensively atheromatous, a condition in itself capable of producing hypertrophy of the left ventricle.\* Finally, in 32 cases out of 48, aneurism of the aorta existed without hypertrophy of the left ventricle, and in 4 of these the ventricle was actually in a state of atrophy. Sir Dominic Corrigan has expressed a very decided opinion on this question. He says: "Aneurism of the arch of the aorta has no tendency to produce enlargement of the heart."† Dr. Stokes declares that "where the heart or its valves have not been previously engaged, there is no reason to believe that the existence of aneurism in any portion of the aorta throws additional labour on the heart, and hence, we commonly find a healthy heart coexisting with a vast aneurism."‡ Professor Axel Key has come to a similar conclusion.§ Walshe, however, believes that aneurism of the aorta, near the heart, is capable of producing hypertrophy of the left ventricle.||

The *diagnosis* of aneurism of the aorta has reference to, (a) its identity, (b) its variety, and (c) its precise seat of origin. The positive determination of these several points, as far as it can be accomplished, constitutes a necessary preliminary to prognosis and treatment. It is, however, in many cases attended with

\* *Vide* Case 101, p. 889.

† *Proceedings of the Pathological Society of Dublin*, February 13th, 1841.

‡ *Diseases of the Heart and Aorta*, p. 579.

§ *Vide* p. 486 for a full discussion of this subject. Since the date at which this passage was written, I have treated three additional cases of aneurism, which are included in the foregoing summary.

|| *Diseases of the Heart and Great Vessels*, *opus citat.*



great, and in a few, with insuperable difficulty. As to the actual existence of aneurism in any given case, suspicion of its presence having been once aroused, a careful scrutiny of the evidence presented will very rarely fail to lead to a positive conclusion.

The *symptoms* or rational signs of aneurism of the aorta may be classified under the two heads of "subjective" and "objective;" whilst the "physical signs," by which is meant the evidence of aneurism, whether proper to the tumor itself, or due to its relations with other parts, discoverable by auscultation, percussion, and palpitation, may be divided accordingly into "intrinsic" and "extrinsic" signs. In practice, however, it will be found more convenient and more instructive to study the disease in its clinical aspect, as presenting definite groups of phenomena, to each of which is assigned a value, determined not only by its special and individual significance, but likewise by its association with other phenomena, whether symptoms or signs.

It will be convenient to discuss, in the abstract, the symptoms and signs which indicate the presence of aneurism in the chest and in the abdomen respectively, before proceeding to consider the modifications which they may present, according to the variety, the point of origin, and the position of the aneurism in both these situations.

The symptoms of aneurism are, for the most part, due to excentric pressure. These may arise, either from the direct pressure of the tumor upon adjacent organs and structures, or from the irritation of parts more or less distant, through the medium of sentient, motor, or vaso-motor nerves.

The pain of aneurism is mainly the result of excentric pressure upon adjacent sentient nerves, and is referred to the seat of their distribution. It is usually shooting, paroxysmal, and of extreme severity, not unfrequently eliciting cries of agony from the unhappy sufferer; it is likewise intermittent. The pain of abdominal aneurism, especially of that which is located behind the crura of the diaphragm, or implicating the solar plexus, is much more severe than that which attends the disease when located in the chest.

The consequences of the mechanical pressure of an aneurism



are not only various, according to the organs affected by it, but, owing to the remarkable proneness of aneurismal tumors to vary in direction and rate of growth, the symptoms arising from the pressure exercised by them upon particular organs and structures are singularly variable and remittent. It is necessary to consider the effects of excentric pressure in the thorax and in the abdomen, respectively.

Thoracic aneurism most frequently tends to the anterior surface of the chest. Hence, it may encroach upon the sternum, the ribs, costal cartilages and intercostal muscles, and the clavicles; absorbing or displacing in its progress such of these parts as it happens to bear upon, and ultimately projecting on the external surface in the form of a yielding and pulsating tumor. The symptoms which attend the advance of an aneurism to the front of the chest are limited to pain of a dull or aching character, extending, not unfrequently, to either shoulder, to the corresponding side of the neck and head, and down the arm, in the course of the cervical and brachial nerves. Severe darting pains of a neuralgic character, radiating round the chest in the course of the intercostal nerves, are likewise occasionally felt even before the tumor has appeared upon the surface. The advance of an aneurism of the lower portion of the arch, or the thoracic aorta, may compromise the left bronchus or lung; obstruction to the circulation of air, or disorganization of the pulmonary structure, being the result. The heart may be dislocated forwards by an aneurism situated low down in the thorax, whilst, by the lateral enlargement of such an aneurism, the œsophagus or the right bronchus may be obstructed or occluded. By the expansion of the sac of an aneurism implicating the anterior wall of the chest, collateral parts may be also pressed upon; hence, frequent obstruction of the descending cava, and venous congestion of the upper portion of the body; hence, likewise, implication of the pulmonary artery, or of either auricular appendix. The tumor may ascend into the neck, and by its pressure upon the innominate, the carotid, or the subclavian arteries, cause arrest, partial or complete, of arterial circulation in the parts supplied by these vessels, with its various consequences; or it may produce venous stasis by compressing the innominate, the internal jugular, or the subclavian veins. Irritation or paresis of the

several nerves at the root of the neck may likewise result from the upward growth of an aneurism of the arch of the aorta. Thus may be explained numbness or paralysis of the upper extremity on the side affected, dilatation or contraction of the pupil on one, or even on both sides, and the various symptoms having reference to the larynx, viz., dysphonia, stridor, aphonia, and spasm. The backward growth of an aneurism springing from the ascending or the transverse portion of the arch of the aorta, may occasion pressure upon the trachea, the right or left bronchus, the cesophagus, the thoracic duct, the sympathetic nerves, the spinal column, or the ribs; whilst an aneurism of the descending portion of the arch, or of the thoracic aorta may, by its growth in the same direction, compress the last mentioned parts only. Finally, an aneurism arising from the root of the aorta, and enlarging mainly in the direction downwards, may implicate any of the chambers of the heart.

Aneurism of the primary portion of the aorta, growing from one or more of the sinuses of Valsalva, has been most amply illustrated by Thurnam,\* who has collected from various sources 18 cases of aneurism in this situation. In 2 of these the aneurism was double, and in 1 it was treble. Thus, there were in these cases 22 aneurisms, of which the right anterior sinus was the seat of the disease in 12 instances, the left anterior sinus in 4, and the posterior sinus in 6. Of the 22 aneurisms, no less than 13 had grown downwards, and projected or opened into the chambers of the heart. This tendency of aneurism connected with the primary portion of the aorta, had been previously noted by Professor Smith,† and by him attributed to the backward pressure exercised upon the column of blood in this portion of the vessel during the systole of the aorta.

The rule is not, however, without many exceptions. Of 7 examples of aneurism of the sinuses of Valsalva collected from the *Proceedings of the Pathological Society of Dublin*, for the eight years 1863-70, 4 had grown downwards, engaging one or other of the chambers of the heart, and 3 had grown upwards.

As regards the special tendency of aneurisms arising from particular sinuses, to establish a communication with definite por-

\* *Medico-Chirurgical Transactions*, vol. xxiii., 1840, page 323.

† *Dublin Journal of Medical and Chemical Science*, vol. ix.

tions of the heart, Dr. Thurnam's conclusions possess some interest; he has found that those which take origin in the anterior and right sinus are the most liable to open into the right chambers, the pulmonary artery, and the pericardium; those of the anterior and left sinus have a special tendency to open into the left chambers, or the pulmonary artery; whilst aneurisms of the posterior sinus are most likely to communicate with either auricle, but especially with the right. Aneurisms arising from the ascending portion of the arch, above the sinuses, have a tendency to open into the pericardium, the descending cava, the pulmonary artery or its right branch, the right pulmonary veins, or either bronchus. According to Dr. Sibson's calculations these aneurisms have ruptured into the pericardium in the proportion of 45 per cent., into the pulmonary artery, 13·5 per cent.; into the right auricle, 8·5 per cent.; and into the right and left ventricle respectively, 5 per cent.\*

Of the 68 cases given in Table XVI., the aneurism had arisen from the ascending portion of the arch in 30 instances, including an example of double aneurism. The direction of growth of the sac was forward in 9 instances, forward and to the right in 4, forward and to the left in 2, forward and upward in 1, forward upward and backward in 1, backward and to the right in 5 (in 4 of which the superior vena cava had been either compressed or opened into), upward and to both sides in 1, downward and to the right and opening into the right ventricle in 1; there was general expansion of the sac in 4 instances.

Innominate aneurism may, by its pressure upon adjacent parts, give rise to extrinsic phenomena identical with those caused by the upward growth of an aneurism of the ascending portion of the arch of the aorta, or of the first half of the transverse portion.

In the abdomen, aneurism of the aorta may, by its backward growth, erode the spinal column, and even penetrate the spinal canal. It may likewise, after perforating the diaphragm, cause absorption of the false ribs, and appear externally in the inferior dorsal region; or it may project on either side, usually the left, between the last rib and the crest of the ilium. By its growth in this direction, an aneurism of the abdominal aorta may like-

\* *Medical Anatomy*, p. 53.



wise make pressure upon either kidney or ureter, the poæ muscles and lumbar nerves. If it advance towards the anterior wall of the abdomen, the pancreas, the liver or bile-ducts, the hollow viscera, or the solar plexus, may be implicated. By its lateral growth, an aneurism within the abdomen may compress or open into the inferior cava, and thereby cause venous stasis in the lower half of the body. It may ascend into the chest after penetrating the diaphragm, and implicate the pleura or lung on either side, or the pericardium. Finally, it may descend into either iliac fossa, or into the true pelvis, involving in its progress the organs and parts lodged in these recesses. The pain caused by the pressure of an abdominal aneurism upon the solar plexus is intermittent, paroxysmal, and of the most intense severity; that connected with the process of gradual absorption of the vertebræ, is dull, persistent, and boring in character, as shown by Dr. Law.\*

The symptoms of thoracic aneurism, although in exceptional instances liable to variation in regard to locality, intensity, and combination, possess a definite value, and, once clearly identified, afford a standpoint whence, by a careful process of exclusion, a positive diagnosis may in most instances be arrived at. But, owing to the rigid character of the thoracic parietes, the great capacity of the chest, and the variable volume of the lungs, the presence of an intra-thoracic tumor, even of considerable magnitude, is not always announced by definite symptoms; and when those which indicate the presence of a tumor are well pronounced, it becomes necessary to determine, in the first instance, whether it is or is not aneurismal.

The symptoms indicative of aneurism within the chest may be now considered in detail. They include pain (intrinsic and extrinsic) visible or palpable tumor and the effects of pressure upon (a) blood vessels, (b) the laryngeal nerves, (c) the trachea, bronchial tubes, or lungs, (d) the œsophagus, (e) the sympathetic or the cardiac nerves, (f) the heart, (g) the thoracic duct, (h) the diaphragm, and (i) the vertebræ.

The intrinsic pain of thoracic aneurism, though not urgent, is not the least significant of its symptoms. It is localized, deep-seated, and constant, oppressive and constrictive rather than sharp, and readily aggravated by causes which quicken the cir-

\* *Dublin Journal of Medical Science*, vols. xxi. and xxii.



culation, and is then associated with dyspnoea in greater or less degree. This pain is caused by tension and subacute inflammation of the sac, and by pressure upon its nerves of supply; hence, the alleviation of it which follows the abstraction of blood or other measures calculated to reduce intra-vascular pressure.

Extrinsic pain, or that due to pressure upon adjacent sentient nerves, is of a totally different character; it is sharp and paroxysmal, ill-defined, wandering, and eminently neuralgic in character; it is usually most severe at night, and, because existing in parts very rarely invaded by neuralgia or muscular rheumatism, namely, the superior dorsal region, neck, walls of chest, and either arm, is of the utmost diagnostic value. The parts affected by these pains may be either benumbed or hyperæsthetic. Where other symptoms suggestive of aneurism likewise exist, the significance of flying pains of this character in the situations mentioned will be proportionately enhanced; but occasionally they constitute the only actual symptom of aneurism,\* and should never be regarded with indifference. The patient speaks of those pains as "rheumatic," and generally connects them with casual exposure to cold or the use of damp clothing, but they are uninfluenced by atmospheric changes, and by anti-rheumatic treatment.

The existence of a pulsating tumor in the course of the aorta at any point of the surface of the chest, but especially on its anterior or posterior aspect, or projecting into the neck, though not amounting to absolute proof, affords stronger evidence of aneurism than any other isolated symptom. The characteristics of an aneurismal tumor are of two kinds; namely, those which belong to it in its simplest form, and those which are incidental to it. The former are: softness, combined with tension and elasticity; decrease of volume under pressure; a strong, heaving, and expansile impulse synchronous with that of the heart; and progressive growth. One or more, or even all, of these features may be replaced by accidental changes in the sac or its contents. Thus, the tumor may have become, in greater or less degree, solid by deposit of fibrin or coagulation of blood within the sac. In such case the qualities of softness and elasticity, yielding un-

\* See Case 139.

der pressure, and systolic expansion of the aneurism, might be greatly modified, or even entirely abolished, and the growth of the tumor arrested.

\* It is further characteristic of an aneurismal tumor appearing upon the exterior of the body, and taking the ordinary course, that, at an advanced stage of its progress, it exhibits upon its surface one or more projections, which are red, tender, and more yielding than the surrounding portions. Here the sac has been thinned or perforated by ulcerative absorption, and its tegumentary or mucous investment is in progress towards sloughing. The retrocession or disappearance of a tumor, previously visible and palpable, whilst common to all those with liquid contents, is characteristic of aneurism as contrasted with cancerous growths, and coincides with the enlargement of the sac at some other point or points of its surface. I cannot acquiesce in the opinion expressed by Dr. Walshe, that cancerous tumors of the mediastinum, no less than those which are aneurismal, may recede within the chest after having appeared externally.\* Dr. Walshe has not adduced an example of such a phenomenon, nor has Dr. Risdon Bennett in his exhaustive treatise.† The appearance of a tumor on the surface of the chest in the course of the aorta, should not only raise a suspicion of aneurism and suggest a careful examination with a view to diagnosis, but the existence of an aneurism having been positively determined, the situation of the tumor may serve to indicate, with more or less precision, the portion of the aorta whence it takes origin.

A tumor unmistakably aneurismal, situate upon the right side of the chest, between the first intercostal space and the third costal cartilage, and inside the nipple-line, would, most probably, be connected with the ascending portion of the arch above the sinuses of Valsalva. Aneurism arising from one or more of the sinuses usually *descends*, and would, therefore, present on the surface, if at all, below the level indicated; whilst innominate aneurisms usually project above that level, and *ascend* into the neck. It is, however, necessary to bear in mind that exceptions to both these statements, especially the former, are occasionally met with. The preponderating frequency of aneurism in this portion of the aorta, as compared with all other portions of it,

\* *Diseases of the Heart*, 4th edition, 1873.    † *Intra Thoracic Growths*, 1864.

and with the arteria innominata, should be duly estimated where doubt exists. An aneurism centred at the right margin of the sternum, the inner portion of the second costal cartilage, and first interspace, has its origin most probably at the first curve of the arch, implicating, or not, the root of the innominate artery; a point to be determined by reference to the circulation in the right carotid and subclavian arteries.

Aneurisms of the innominata rarely point at this level, and still more rarely fail to appear, at an early stage, above the clavicle. The downward extension of percussion-dulness, and the diffusion of impulse in the same direction, would, in such a case, be in favour of the diagnosis of aortic, and against that of innominate aneurism.

A pulsating tumor, having its point of greatest prominence at the centre of the *manubrium sterni*, especially if an impulse were perceptible above the fourchette, and venous congestion existed at the root of the neck, would indicate aneurism of the transverse portion of the arch somewhere about its centre; whilst aneurism of the left extremity of this portion of the vessel would be most likely to point beneath the left clavicle. In a case observed by Dr. Mayne, aneurism of the ascending portion of the arch had pointed to the left of the sternum, making pressure upon the left bronchus;\* and in another, recorded by Dr. Greene, an aneurism of the descending aorta had passed over to the right side behind the œsophagus, and compressed the right bronchus.†

Aneurisms of the descending portion of the arch, or of the thoracic aorta, pointing externally, most frequently appear in the left scapular, or infra-scapular region; they may, however, project above the left clavicle.

Diminished force, or even total suppression of circulation in adjacent arteries, or in those arising from the sac, may be caused by the excentric pressure of an aneurism of the aorta, by the formation of an autochthonous clot, the impaction of one detached from the sac, or by the extension of the contained coagula

\* *Proceedings of the Pathological Society*, December 20th, 1851; see also a case by Dr. T. Evelyn Little, *ibid.*, vol. v., part ii.

† *Dublin Journal*, vol. vii., 1835.

over the mouth of the vessel. Between these several causes of obstructed arterial circulation, the presence or absence of a pulsating tumor, and its position with regard to the obstructed vessels, must serve as the principal means of distinction. Where no tumor exists, the probability of endarteritis with plugging might be inferred. Mahomed declares that the pulse-tracings exhibit no special characters distinctive of obstruction from these several causes.\*

I have observed postponement of the radial pulse, first noticed by Henderson, as a sign of aneurism of the arch, in one case (146) only.

Partial suppression of pulse in the carotid or subclavian artery, or in both these vessels, on one side of the body, may be likewise due to the pressure of a mass of enlarged lymphatic glands in the anterior mediastinum or in the neck.† In connexion with thoracic aneurism, these phenomena are not unfrequently exhibited in the carotid or subclavian artery of either side, and occasionally in both these vessels on one side of the body. Complete occlusion of the common carotid and vertebral artery on one side, has been followed by hemiplegia from anæmia and atrophy of that side of the brain. The existence of obstruction in the vessels of *both* sides is characteristic rather of cancerous tumor than of aneurism. Inequality of radial pulsation may be readily detected by the finger, but the crucial test is supplied by the use of the sphygmograph. It is noteworthy that, in arteries obstructed by an aneurism, whilst the usual progress leads gradually to permanent suppression of pulsation, the pulse may exhibit considerable variations of volume within a brief period; it may be even temporarily suspended, and, by removal of external pressure from the vessel, subsequently restored to its normal condition.‡ Such variation in the pulse, where attributable to a tumor, is eminently suggestive of aneurism.

The pressure of an aneurism upon the great veins within the chest is indicated by venous turgescence, with œdema, of the portions of the body whence blood is ordinarily returned to the heart through the compressed vein. Congestion and œdema of the right side of the head and neck only, with turgescence of

\* *Medical Times and Gazette*, August 30th, 1873.

† *Vide* Case 71, p. 736.

‡ See Case 142.



the external jugular vein, would indicate pressure upon the corresponding internal jugular, and if the compressing body were an aneurism, its seat would most probably be the innominate or the common carotid artery. On the left side, congestion similarly limited would indicate the pressure of a carotid aneurism. Localized congestion and œdema of one upper extremity from the pressure of an aneurism, would point to the subclavian vein as the subject of compression and the subclavian artery as the seat of the disease. Simultaneous congestion of all these parts on one side would indicate pressure upon the corresponding innominate vein, from an aneurism of the transverse portion of the arch, if on the left side, and of the innominate artery on the right. Congestion and œdema, limited to the upper half of the body, and depending upon the pressure of an aneurism, would indicate the ascending portion of the arch, or the right extremity of its transverse portion, as the seat of the disease.

The symptoms produced by the pressure of an aneurism upon the trachea, the bronchi, the lungs, or the recurrent nerves, are remarkably various, and of the utmost diagnostic value. It occasionally happens that, with the exception of the symptoms so produced, no others decidedly suggestive of tumor within the chest, can be detected.\* Yet, so characteristic are they, that, when duly estimated, they constitute in themselves evidence sufficient to warrant a positive diagnosis of intra-thoracic tumor, the nature of which must be determined by collateral symptoms and signs.

The symptoms referable to the larynx may be first considered. These are the result either of spasm or paralysis of the intrinsic muscles of the larynx, affecting one side only, or both sides, from the pressure of an aneurism upon the pneumogastric or the recurrent nerves. Spasm of the larynx is most frequently unilateral, and a consequence of partial pressure upon the recurrent laryngeal nerve of the same side; it is, however, very rarely unaccompanied by the symptoms of tracheal pressure. It is characterized by paroxysms of the most urgent dyspnœa, accompanied by laryngeal stridor, and usually brought on by exertion or by excitement of any kind. During the paroxysm, the features are haggard and expressive of great alarm, the pupils are

\* *Vide* Case 133; also a case reported by Drs. O'Ferrall and Stokes, in the *Proceedings of the Pathological Society of Dublin*, March 24th, 1860.

dilated, the face more or less congested and of a leaden hue, the nostrils are dilated, and the muscles of the chest and shoulders in a state of tension during the struggle for breath. The action of the heart is tumultuous, the carotid and subclavian arteries bound visibly, whilst the radial pulse is quick, small, and hard. The agony may last for several hours. It not unfrequently terminates in syncope, from which the patient gradually recovers, and is then quite free from suffering till the occurrence of the next paroxysm; or death may be the result of one of these seizures.\* During the interval, which is of uncertain duration, the voice is unaffected, and there may be a total exemption from symptoms; but, usually, there is more or less difficulty of breathing. Respiration is readily quickened by exercise, and then the patient has harsh and dry cough of a sharp and metallic character. As pointed out by Dr. George Johnson, the stridor is most distinctly audible, during a paroxysm, over the middle cervical vertebræ,† and laryngoscopic inspection during the intervals will show that the larynx is in its normal condition.‡

More decided pressure upon the pneumogastric or recurrent nerve of either side, will cause paralysis of the laryngeal muscles. This is usually limited to the side affected; but in two cases reported, one by Dr. Bäumlér,§ and another by Dr. Johnson,|| bilateral paralysis of the larynx resulted from pressure upon the pneumogastric and recurrent nerves of one side only.¶ When laryngeal symptoms are the result of paralysis, there is either aphonia or permanent huskiness of voice, and if an ex-

\* *Vide* the case of the late Earl St. Maur, by Dr. C. J. B. Williams, pamphlet, 1870.

† *British Medical Journal*, May 23rd, 1874.

‡ See case 138.

§ *Transactions of the Pathological Society of London*, vol. xxiii., p. 66.

|| *Ibid.* vol. xxiv., p. 42.

¶ In a more recent communication (*British Medical Journal*, December 19th, 1874), Dr. Johnson has expressed the opinion, that in these and all similar cases, paralysis of the laryngeal muscles of the opposite side is due to a reflex influence conducted to the opposite recurrent nerve, through the commissural connexion between the spinal accessory nuclei. I cannot conceive how reflex paralysis of the opposite recurrent nerve, which must be necessarily *partial*, as influencing only the fibres of origin on the side of pressure, could produce *complete* paralysis of the muscles supplied by it. No such difficulty exists in regard to the theory of reflex spasm.

mination be made with the laryngoscope, the arytenoid cartilage and vocal chord of one side, or those of both sides, will be found immovable during forced inspiration and the attempt to vocalize, whilst the larynx is obviously free from congestion and the other usual causes of hoarseness. As in ordinary cases of paralysis, the muscles of the larynx, if long paralysed, become wasted. Dr. Morell Mackenzie has published two cases of aneurism of the arch of the aorta, causing paralysis of the muscles of the left side of the larynx by pressure upon the left recurrent nerve. In one of these cases, which was of eighteen months' duration, the paralysed muscles had undergone atrophy.\* Aneurisms which give rise to laryngeal symptoms by pressure upon the pneumogastric or recurrent nerves exclusively, most frequently arise from the upper extremity of the ascending, or the transverse portion of the arch, and from the anterior or the superior surface of the vessel; hence, they are announced by visible tumor, perceptible pulsation, venous congestion in the neck, and other palpable evidence of their existence. The affected nerves are always upon the stretch by the expansion of the sac, and not unfrequently they are flattened upon, and incorporated with it.

Pressure upon the trachea may result from an aneurism arising from any portion of the arch of the aorta, or from the arteria innominata. When produced by an aneurism of the ascending portion of the arch, or by one connected with the anterior or the superior surface of its transverse portion, the symptoms of pressure upon the trachea are *late* in the order of events, and are usually associated with those of irritation or paralysis of the laryngeal nerves, and with dysphagia. Such is likewise the case in regard to aneurisms of the descending portion of the vessel. The most urgent symptoms of tracheal pressure are, however, in many cases, produced by an aneurism springing from the posterior surface of the transverse portion of the arch. In such connexion, they are, moreover, of early occurrence in the history of the case, and sometimes constitute the first, and, for a long period, the only symptom of the disease. Although the symptoms of tracheal pressure constitute the most salient feature in many cases of aneurism, they are, in my opinion, very

\* *British Medical Journal*, May 21st, 1870.



rarely unassociated with those of laryngeal spasm, and both may be produced by an aneurism not implicating the recurrent nerves.\* The tracheal and bronchial nerves can scarcely escape being involved, and reflex irritation through these nerve-filaments, intimately associated as they are with the pneumogastric and its branches, will account for the usual association of laryngeal symptoms with those of tracheal pressure.

The trachea, when pressed upon by an aneurism, is flattened and more or less indented upon one side, and, when the pressure is extreme, stretched and bent towards the opposite side. When the trachea is laid open, its lateral wall is seen to bulge inwards at the point of pressure, and, if the cartilages have been already eroded, the mucous membrane in that situation is congested, of a deep purple colour, or perhaps in a state of incipient gangrene preparatory to irruption of blood from the sac. Owing to the peculiar conformation of the trachea, and the laxity of its connexions, it readily eludes pressure from the front, moving to either side according to the inclination of the impinging body, and receiving the impression upon the side opposite. The trachea, when compressed by an aneurism arising from the ascending portion of the arch, from the right extremity of its transverse portion, or from the arteria innominata, receives the pressure upon its right surface; but when the aneurism arises from the left portion of the arch, whether transverse or descending, the trachea, if encroached upon by the sac, will receive the pressure upon its left side.

Owing to the relative positions of the trachea and the arch of the aorta, whence compressing aneurisms most frequently spring, the portion of the former usually pressed upon by an aneurism is that immediately above the bifurcation. The symptoms and signs which indicate pressure upon the trachea, not implicating the larynx, are the following: Dyspnœa, with a feeling of tightness and obstruction behind the upper portion of the sternum; stridulous inspiration, the stridor being loudest over the superior dorsal spine, as indicated by Dr. Johnson, or "from below," as long since noted by Dr. Stokes; a dry and harsh cough, with a decided metallic clangour; and voice of a muffled or distant and ventriloquial character. These phenomena become more significant if intensified by exertion or excitement. Facial

\* See Case 138.



decubitus, and frequent extension of the neck, have been noted by Dr. Bellingham and Dr. Law, respectively. Examination by means of the laryngoscope will show that the larynx is unaffected, the glottis expanding fully with inspiration, and voice of the character above mentioned being readily educed. There is, in most cases, some inequality of respiration on the opposite sides of the chest, the vesicular murmur being less distinct on the side corresponding to that of pressure, and associated with tracheal breathing and voice at the upper part of the sternum and in the interscapular space on the same side. This is usually the result of pressure upon the main bronchus, but it may be caused by pressure upon the trachea exclusively. A tumor projecting into the trachea close to the orifice of either bronchus, may divert the current of air in some degree to the opposite side, and thus become the cause of unequal inflation of the lungs; whilst the implication of the bronchial nerves in the sac may excite spasm, and lead to the same result. In Case 138 the inequality of respiration would seem to be explicable only on one or both of the foregoing hypotheses. Suppression or decided feebleness of respiration in one lung, if persistent, would indicate mechanical obstruction of the bronchus of that side; and, if associated with the vocal and other phenomena above mentioned, engagement of the trachea might be likewise inferred. Aphonia cannot be included amongst the ordinary symptoms of tracheal or bronchial compression; in one case, however, observed by Professor Smith, and referred to by Dr. Stokes,\* in which the trachea alone was implicated, aphonia existed without stridor.

Where the symptoms of laryngeal spasm are superadded to those of tracheal compression, as they usually are towards the termination of every case in which the trachea is implicated by an aneurism, the most urgent respiratory distress, not unfrequently ending in death by asphyxia, is exhibited. The larynx is rarely altered in structure, but in one such case (No. 142) well marked cedema of the glottis existed, most probably the result of venous congestion from pressure.

The symptoms produced by the pressure of an aneurism upon one of the bronchial tubes, without implicating the trachea, are few and indefinite previous to irruption of blood from the sac;

\* *Opus citat.*

not so, however, the signs discoverable by auscultation and percussion. The left bronchus is more frequently the subject of pressure than the right, owing to its anatomical relationship to the descending portion of the arch and the thoracic aorta. The greater number of aneurisms within the chest tend to advance towards its anterior wall, and those connected with the ascending and transverse portions of the arch, which, by their backward growth, involve the right bronchus, generally implicate the trachea also. Hence, isolated bronchial pressure is most frequently witnessed on the left side. There may be defective expansion of the affected side of the chest, owing to imperfect inflation of the lung supplied with air by the obstructed bronchus, but so many causes, having reference to actual and antecedent conditions of the lung, may give rise to this symptom, that, apart from other evidence, it must be regarded as of little practical value for the purpose of diagnosis. The degree in which it is exhibited where obstruction of a bronchus, not amounting to complete occlusion, exists, is, moreover, very trivial, and by no means readily detected. The method proposed by Sir Dominic Corrigan,\* whether for the purpose of detecting imperfect expansion or deep-seated pulsation at any point of the surface of the chest, is by far the most delicate and trustworthy. This consists in placing the patient in a strong light, the chest having been entirely denuded, and then looking downwards obliquely from behind the shoulder on the affected side.

Complete occlusion of a bronchus by the pressure of an aneurism is exceedingly rare. Dr. Stokes declares he has never met with an example of it.

Cough is another and a constant symptom of bronchial pressure. It is generally associated with bronchitis, and hence, regarded separately, it avails very little as evidence of aneurism or other tumor within the chest. Owing to pressure upon the pulmonary plexus the cough may partake of the ringing or aneurismal character, and when this is the case, it possesses a decided and positive significance. Occasionally the mucus expectorated is streaked with blood, and there may be veritable hæmoptysis from local congestion, or from leakage of the aneurism.

Doctor Greene† has observed retraction of the trachea with

\* *Lancet*, February 7th, 1829.    † *Dublin Journal of Medical Science*, vol. vii.

inspiration, in two cases of aneurism pressing on the left bronchus. Where the primary bronchus of either side is compressed by a tumor, the respiratory murmur and the vocal fremitus are proportionately diminished in the lung supplied by it; whilst in the opposite lung these phenomena are increased in an inverse ratio. Occasionally, also, as noticed by Dr. Greene and Dr. Stokes, air is heard to enter the affected bronchus "with a rush" at the acme of a forced inspiration. Inequality of respiratory sound is frequently noticeable, as between the upper and the lower portions of the lung affected by pressure upon its main bronchus. Respiration is, in such cases, usually more feeble in the apex than in the base of the lung, a difference which I believe to be due to pressure also upon the secondary bronchus through which the apex of the lung is inflated.\* With defect of respiratory sound in either lung, are usually associated bronchial respiration and bronchophony in the scapular region, or the interscapular space of that side, whilst vocal thrill is imperfect, and may even be abolished, over those portions of the lung in which respiration is most enfeebled. With the signs above mentioned a clear sound on percussion is associated, where consolidation of the lung or pleuritic effusion does not exist. Amongst the consequences of bronchial obstruction by pressure *ab extra*, congestive pneumonia and gangrene of the lung must be included. Pneumonia of a low or asthenic character, and readily passing into gangrene, is a frequent result of the pressure of an aneurism upon the bronchial vessels and nerves, as shown by Carswell.† It is usually localized, and where considerable impediment to the entrance of air into the affected portion of the lung, by pressure upon the bronchus, also exists, it is distinguished from all other forms of pneumonia by the absence of vocal fremitus.‡ Pneumonia, characterized by the ordinary signs, may, however, result from the implication of the pulmonary structure by the direct pressure of an aneurism. In the progress of such cases, repeated hæmoptysis from leakage of the aneurism, or from rupture of the pulmonary capillaries, is a usual symptom.§

\* *Vide* Cases 141 and 142. † *Pathological Anatomy*. ‡ *Vide* Case 136.

§ See two cases by Dr. Mayne, *Proceedings of the Pathological Society of Dublin*, April 6th, 1861, and January 4th, 1863.



Feeble respiration with percussion-resonance on one side of the chest may, however, be due to the following causes, irrespectively of aneurism: local thickening of the bronchial membrane and consequent obstruction, as pointed out by Andral;\* local emphysema; the impaction of a foreign body in the bronchus, a cancerous tumor, or enlarged bronchial glands pressing on the bronchus. Dr. Greene has shown that not only feeble respiration in one lung, but dyspnoea, dysphonia, stridulous breathing, and violent spasmodic and ringing cough, may result from the pressure of enlarged bronchial glands upon one of the bronchi.†

Doctor Stokes has published a case in which all the foregoing symptoms, except dysphonia, together with dysphagia and most of the physical signs of aneurism, were produced by a large cancerous tumor occupying the posterior mediastinum.‡

The distinction between these several causes of unequal respiration on the opposite sides of the chest, must rest upon collateral evidence. Local bronchitis is exceedingly rare; indeed, it is characteristic of the bronchial membrane that the changes induced by local irritation are rapidly diffused over its entire tract. The detection of local emphysema by means of its proper signs cannot be attended with much difficulty; the prolonged expiratory wheeze and exaggerated percussion-resonance with which it is accompanied, will suffice to distinguish it.

The diagnosis of bronchial pressure caused by a cancerous tumor is often extremely difficult, and sometimes impossible. When growths exhibiting the characters of scirrhus or encephaloid cancer exist anywhere upon the exterior of the body, or in situations where their nature can be determined, a presumption in favour of internal cancer, and against the presence of aneurism, would be warranted. The persistency of pressure would be in favour of the same view, whilst the physical signs peculiar to both these diseases, to be discussed presently, would afford still more important aid towards the distinction. In the case by Dr. Stokes, above referred to, a single systolic murmur existed. This sign, in connexion with a tumor in the posterior mediastinum, was suggestive of cancer rather than of aneurism.

\* *Clinical Medicine*, vol. i., Observation ii.

† *Dublin Journal of Medical Science*, vol. x., 1837.

‡ *Ibid.*, vol. xxi.



The obstruction resulting from the lodgment of a foreign body in one of the bronchial tubes is usually more complete than that produced by the pressure of an aneurism. Hæmoptysis and local inflammation are quickly induced by its presence, and, contrary to what obtains where aneurism is the cause of obstruction, respiration is *equally diminished over the entire lung*. Finally, the history of a misadventure in which a foreign body *may* have found entrance into the air passages, and the absence, previous to such occurrence, of all symptoms pointing to aneurism, would be, in themselves, almost conclusive as to the cause of obstruction.

Dysphagia is a very common symptom of thoracic aneurism. It may be due to mechanical obstruction from the direct pressure of the tumor upon the œsophagus, which is the ordinary mode of its occurrence; to spasm of the gullet from direct or reflex irritation of the œsophageal plexus; or to paralysis of the muscular wall of the œsophagus from destruction of its motor nerves, or from excessive dilatation.

Doctor Davy, of Terenure, has recently published a most instructive case of dysphagia from dilatation of the œsophagus, in which, in addition, many of the more specific symptoms of aneurism were exhibited; *e.g.*, epigastric pulsation with tenderness and percussion-dulness, pains in the back and shoulder, and a "tearing or raking" sensation at the epigastrium on attempting to swallow. Deglutition was, moreover, less difficult whilst he lay on the right side; and all these symptoms gradually followed a strain in lifting a weight ten years previously. There was no hæmatemesis or enlargement of the external glands. Death was ultimately caused by inanition, and, on examining the body, the œsophagus was found to be enormously dilated, measuring nine inches in circumference some distance from its termination, and eight inches above and below this point; it was capable of containing forty ounces of liquid. The muscular coat was hypertrophied, and, in the most dilated portion, the mucous membrane presented several recent ulcers, and a still greater number of cicatrices. The œsophagus presented a twist from right to left, where it passed through the diaphragm. This was, no doubt, the cause of dilatation and all the subsequent symptoms. The stomach was dilated and its walls were thinned; the pylorus was

healthy.\* In this case it would have been impossible during life to affirm that aneurism did not exist.

Aneurisms of the transverse and descending portion of the arch, and of the thoracic aorta, are those which most frequently make pressure upon the œsophagus. An aneurism of the ascending aorta may, however, produce this effect. The late Dr. Eames presented to the Pathological Society of Dublin an example of the latter kind. Three aneurisms were connected with the ascending portion of the arch of the aorta, and of these, the lower arose from the posterior sinus of Valsalva, and, passing backwards, pressed upon the œsophagus, and caused dysphagia with boring pain in the back.† Dysphagia resulting from the direct pressure of an aneurism is usually remittent; it may even entirely cease, and return after a long interval.‡ It may likewise vary in site, as shown by Dr. Law,§ and, as noted by the same observer, it may exist in the recumbent posture only. The patient can, in most cases, swallow liquids with facility, but deglutition of solids is attended with pain referred to the upper, middle, or lower portion of the sternum, according to the seat of obstruction. The difficulty of swallowing has been occasionally overcome by varying the posture of the body whilst making the effort, a circumstance which might be regarded as conclusive evidence of obstruction of the œsophagus either by the pressure of a tumor, the nature of which must be determined by reference to collateral symptoms, or by torsion of the tube itself, as in the case given by Dr. Davy.

Simple pressure upon the œsophagus, in the absence of a resisting point of counter pressure, ingrowth of the sac, or irritation of the muscular tunics or motor nerves by local inflammation, is not attended with dysphagia. Hence, displacement of the œsophagus to either side is not necessarily followed by symptoms of obstructed deglutition. Owing to the laxity of its connexions, the yielding character of its structures, and its great elasticity, the œsophagus may undergo considerable displace-

\* *Irish Hospital Gazette*, May 1st, 1875.

† Meeting of March 5th, 1870.

‡ *Vide Stokes, Diseases of the Heart and Aorta*, Case 72, p. 576.

§ *Proceedings of Pathological Society*, February, 1841.

ment into either pleural cavity, under the pressure of a smooth-surfaced tumor, without suffering impairment of function.\*

Dysphagia from spasm is most frequently referred to the scrobiculus cordis. It is characterised, as spasm usually is, by suddenness of development and cessation, and by being provoked by *dry* rather than by *moist* solids. A feeling of sharp pain in a particular part of the gullet, at the moment of passage of the alimentary bolus at that point, in a case of dysphagia from the pressure of an aneurism, would indicate erosion of the mucous membrane and early communication with the sac by ulceration or sloughing.

Contraction of the pupil from paralysis of the cervical sympathetic was first observed by Pourfour du Petit, who likewise noticed, in connexion therewith, drooping of the upper lid, and cedema of the eye. Paralysis was induced by section of the nerve, and the observation, though made in 1712, was not published till 1727.† Dupuy, early in the present century, noticed the same results from division of the sympathetic nerve; but he further observed increased temperature of the ears, perspiration, and eruptions on the skin.‡ Dr. John Reid was the first who showed that contraction might result from pressure on the sympathetic.§ Biffi, on the other hand, noticed dilatation of the pupil as a result of galvanizing the upper end of the divided nerve.|| Bernard¶ and Brown-Séquard\*\* confirmed the observations of Dupuy, and further extended our knowledge, by proving experimentally that, not only alterations of the pupil, but likewise decided modifications of vascularity, temperature, and sensibility, may result from irritation or paralysis of the sympathetic in the neck. About the same time Valentin pointed out that the nerve endowment of the iris was twofold, its radiating fibres or dilator muscle receiving filaments from the

\* *Vide* Case 140, and Fig. lxxiv.

† *Mem. de l'Acad. des Sc. de Paris*, 1727, quoted by Longet, *Traité de Physiologie*, 3<sup>me</sup> edit., 1869, tom. ii., p. 932.

‡ *Jour. de Méd. Chirurg. et Pharm.*, Décembre, 1816.

§ *Edinburgh Medical and Surgical Journal*, January, 1838, and August, 1839.

|| *Annali Universali di Medicina*, 1845, Longet. *Ibid.*

¶ *Comptes Rendus de l'Académie des Sciences*, Mars, 1852.

\*\* *Mém. lu à l'Acad. des Sciences*, January, 1854.

sympathetic, and the circular fibres or sphincter, from the oculomotor nerve. He also showed that the ciliary fibres of the sympathetic were derived, primarily, from the spinal cord.\* Budge and Waller about this date also, proved by a series of experiments, that the sympathetic nerve-filaments of the iris take origin from the portion of the spinal cord between the sixth cervical and the fourth dorsal vertebræ, which portion they accordingly proposed to designate as the "cilio-spinal" region.†

In 1838, Dr. Hare made what may be regarded as the first clinical observation bearing on this subject. He noticed contraction of the pupil in connexion with pressure upon the sympathetic from a scirrhus tumor in the neck,‡ and in 1850, Dr. McDonnell, of Montreal, observed both contraction of the pupils and ptosis from the pressure of a malignant tumor in the same situation.§ The first case in which contraction of the pupil was observed from the pressure of an aneurism was one published by Dr. Walshe.|| Dr. Gairdner's cases were next in order of time,¶ and, in the following year, a case in which perspiration, limited to the side of the face on which the pupil was contracted, was published by the same observer.\*\*

In the same year in which Dr. Gairdner's memoir appeared, Dr. Banks recorded an example of a similar kind.†† In this case, in addition to contraction of the pupil, partial ptosis existed. It is unnecessary to pursue further the history of this remarkable discovery. Since the last mentioned date, the examples of derangement of the pupil from the pressure of an aneurism upon the sympathetic chord in the neck, recorded in the annals of medicine, have been very numerous, Dr. J. W. Ogle has collected from various sources twelve such cases.‡‡ I have myself observed several cases of the kind. The pupil is

\* *De Functionibus Nervorum Cerebraliū*, Bernæ, 1852.

† *Vierordt's Archiv. für Physiol. Heilkunde*, 1852.

‡ *Medical Gazette*, 1838.

§ Quoted by Gairdner from *The Montreal Medical Chronicle* for 1853.

|| *Diseases of the Lungs, Heart, and Aorta*, second edition, 1853.

¶ *Edinburgh Monthly Journal*, August, 1855.

\*\* *Ibid.*, 1856.

†† *Dublin Hospital Gazette*, vol. ii., 1855.

‡‡ *Medico-Chirurgical Transactions*. vol. xli.



not insensible to light; it varies in diameter according to the degree of exposure; but it is always less affected by light or shadow than the pupil of the other eye, nor is sight in any degree impaired. For many of the facts and references in the foregoing *résumé*, I am indebted to the learned author of the exhaustive memoir last referred to (Dr. Ogle).

Constriction of the pupil on both sides may result from bilateral compression of the sympathetic. Of this phenomenon, which is exceedingly rare, an example has been observed by Dr. Gairdner, as a consequence of the extension of a large aneurism of the descending and thoracic aorta to both sides of the vertebral column.

As might have been anticipated from the recorded results of section of the sympathetic in animals, elevation of temperature, local perspiration, ptosis, and internal strabismus, have been repeatedly observed in connexion with contracted pupil from pressure on the sympathetic. The first and the third phenomena were exhibited in Case 142. Dilatation of the blood-vessels from paralysis of the vaso-motor nerves will serve to explain the rise of temperature and sweating. Ptosis and convergent squint, the latter of less frequent occurrence than the former, which is always incomplete, may, as suggested by Dr. Ogle,\* be rationally referred to the double innervation of the levator palpebræ and abductor oculi muscles, through the filaments of communication from the sympathetic to the third and sixth cerebral nerves in the cavernous sinus. One of these two sources of nerve-supply being cut off by paralysis of the sympathetic, the muscles named lose their involuntary functions, whilst remaining under the control of the will. Hence, drooping of the upper lid, and arrest of nictitation, with continued power of opening the eye by a voluntary effort. Dilatation of the pupil has been likewise, but less frequently, observed in connexion with pressure upon the sympathetic; it arises from *irritation* of the nerve, and therefore indicates a less degree of pressure. Dr. Ogle has recorded 7 examples of this phenomenon, viz., 3 as the result of aneurism, 2 from scirrhus growths, one from the pressure of enlarged lymphatic glands,

\* *Loco citat.*

and one from ecchymosis of the cervical portion of the spinal cord. In the two last mentioned cases, both pupils were dilated. Contraction has been observed to supervene upon dilatation of the pupil, and, in one case, to alternate with it.\* Both contraction and dilatation of the pupil, with the attendant phenomena above mentioned, have been likewise observed in connexion with injury of the cilio-spinal region of the cord.†

Modifications of the pupil, in connexion with intra-thoracic or cervical tumor, furnish evidence of nerve-pressure only. The value of this symptom, as a diagnostic of aneurism, is rather relative than absolute, and must be determined by collateral evidence. Associated with any of the symptoms or signs of aneurism, changes of the pupil possess a positive value, not only as contributory evidence of the existence of that disease, but as indicating its locality, and in some degree, the dimensions of the sac.

Doctor Gairdner believes that symptoms of an anginal character may result from pressure upon the cardiac plexus by an aneurism close to the heart.‡ Such symptoms, however, apart from those of paroxysmal dyspnoea from laryngeal and bronchial spasm, which are necessarily accompanied by cardiac excitement, are very rare in connexion with aneurism. Another symptom, however, of kindred character, is occasionally witnessed in cases of aneurism of the aorta, namely, rhythmical irregularity of breathing, or "Cheyne Stokes' respiration."§ I have already, at p. 626, discussed this remarkable phenomenon, and at p. 632, propounded, with diffidence, a theory of its development.

Von Dusch, in 1867, declared that he had observed this symptom in certain forms of cerebral disease, and in a severe case of pericarditis. Traube, four years later, alleged that he had witnessed it in connexion with valvular disease at the aortic and mitral orifices, the heart not being fatty; and likewise in cases of cerebral tumors and hæmorrhage, uræmic coma, and tubercular meningitis. He regards the symptom as due to want of arterial

\* *Vide* case by Dr. Kidd, quoted in Dr. Ogle's memoir.

† *Vide* Ogle, *loco citat.*

‡ *Clinical Medicine*, 1862, p. 495.

§ *Traube*, 1871.

blood in the respiratory nerve-centres, in consequence of which, respiration failing, and carbonic acid accumulating in the lungs, the pneumogastric nerves, and subsequently the sentient nerves generally, are stimulated to inordinate action, and accelerated respiration is the result. But, carbonic acid being rapidly eliminated under this increased activity of respiration, the peripheral sentient nerves, and then the pneumogastrics, are inadequately stimulated; hence, a gradual subsidence of respiration, and finally apncea.

Filehne is of opinion that this symptom arises from a lowering of excitability in the respiratory nerve-centre, relatively to that of the vaso-motor centre.\*

This form of respiratory derangement is unique. I have never witnessed it save in connexion with an advanced degree of atheromatous change and dilatation of the arch of the aorta, with or without fatty degeneration of the heart; and, judging from my personal experience, such as it has been, I feel bound to regard it as pathognomonic of the conditions of the aorta just mentioned.

Obstruction of the thoracic duct by the pressure of an aneurism has been very rarely witnessed. Laennec has recorded one such example, in which lymphatic engorgement of the lower half of the body was a prominent symptom.† The symptoms arising from erosion of the vertebræ are, on the contrary, of frequent occurrence. Such symptoms are most frequently witnessed in connexion with aneurisms of the descending portion of the arch, the thoracic, and the abdominal aorta. Many examples of absorption of the vertebræ by the pressure of an aneurism of the ascending or transverse portion of the arch, have, however, been recorded; but in these cases pressure upon the intermediate parts, the trachea, the œsophagus, and the great nerve-trunks, necessarily preceded, and gave rise to corresponding symptoms.

Erosion of the vertebræ is accompanied by a fixed "boring" pain. With this are usually associated severe radiating pains of a darting or neuralgic character, but intermittent, and referred

\* *Vide résumé in The Irish Hospital Gazette*, November 2nd, 1874.

† *Traité de l'Auscultation Médiate*, tom. ii., p. 714; see also a case by Dr. Hughes Bennett, *Principles and Practice of Medicine*, second edition, p. 567.

to the seat of fixed pain as their centre. The former is due to the action of the tumor upon the proper structures of the vertebral column, and the latter to the spinal compression of nerves, the paroxysms being caused by the temporary expansion of the sac under increased vascular pressure. Spasm, or paralysis of the muscles supplied from the portion of the cord below the seat of pressure, may result from erosion of the vertebra, by irritation or destruction of the corresponding motor nerves. Examples of paraplegia from the irruption of an aneurism of the descending aorta into the spinal canal, have been recorded by Laennec\* and Andral;† and another by Dr. Hughes Bennett, from pressure of the sac upon the spinal cord.‡

The sac, pressed against the solid bone, is quickly broken through by the repeated impulse of the blood, the periosteum is next removed by absorption, and, under the remittent pressure of the aneurism, the osseous texture is starved by interruption of its arterial supply, and ultimately disintegrated by a process of molecular necrosis; the *débris* of the bone-substance is mixed with the contents of the sac. The intervertebral fibro-cartilages, owing to their resiliency, are capable of appropriating sufficient pabulum to maintain their vitality, during the brief periods of interrupted pressure corresponding to ventricular diastole.

The *physical signs* of thoracic aneurism may be conveniently classified under the two heads of extrinsic and intrinsic phenomena. The extrinsic signs, which are identical with those of pressure upon the air passages, the lungs, and the laryngeal and bronchial nerves, have been already discussed. The intrinsic signs of aneurism may be now considered under the heads of (a) percussion-dulness, (b) impulse, (c) sounds, and (d) thrill.

Dulness on percussion is clinically associated with internal aneurism, only when it has come into contact with the thoracic or abdominal parietes. It is absolute, coextensive with the area of contact, and accompanied by a feeling of resistance communicated to the finger. The extent and the degree of dulness are usually progressive; but, owing to retrocession of the tumor in the course of the disease, percussion resonance may replace dul-

\* *Ibid.*, p. 715.† *Acad. de Méd.*, 1854.‡ *Opus citat.*, p. 572.



ness. Not only the area, but likewise the location of dulness, may undergo change from the same cause.

The impulse of thoracic aneurism is most frequently single and systolic. In a somewhat less numerical proportion it is double (systolic and diastolic), as first pointed out by Dr. Stokes in 1834.\* The systolic or principal impulse is heaving and expansile in character, and coincides with the impulse of the heart; the second is abrupt and non-expansile, diastolic or postdiastolic in rhythm, and communicates to the hand placed over the tumor, a sensation as of sudden arrest to the collapse of the sac towards its termination. From this latter peculiarity it has been designated a "back-stroke;" but as this title implies re-expansion of the sac, it is objectionable. A better designation, because more accurately expressive of the cause and character of the phenomenon, would be an *impulse of arrest*, under which title I shall henceforward refer to it. With the systolic impulse, and coinciding with it in time, is not unfrequently associated fremitus or thrill of a more or less decided character.

The systolic or principal impulse of aneurism is due to the forced expansion of the sac, under the pressure of influx from the main artery at the moment of ventricular systole; hence, it is essentially expansile in character. Thrill is by no means an ordinary accompaniment of aneurismal impulse; when present, it is due to vibration of the sac, either from an eddy in the current of influx, or from a lax state of the sac itself. A spiculated condition of the orifice of the aneurism, or a flake of fibrin partially detached from its walls and floating in the passage, may produce a vortex in the blood-stream, and consequent thrill. The existence of tactile fremitus, therefore, not communicated from the heart, is pathognomonic of vascular tumor of some kind, and where independent evidence of aneurism exists, it may be regarded as in the highest degree confirmatory.

Doctor Bellingham regarded aneurismal *frémissement* as due to regurgitation of blood into the sac during the diastole of the ventricle. That this explanation cannot be admitted, will appear from the fact that the phenomenon in question is *systolic*, not diastolic, in rhythm.

\* *Dublin Journal of Medical Science*, vol. v.

The second or diastolic impulse is not of constant occurrence in aneurism, but when present, it is, no less than fremitus, diagnostic of that disease, owing to the peculiar circumstances under which alone it can be produced.

I think Gendrin's explanation of this phenomenon the most rational.\* He refers it to want of synchronism between the reaction of the sac and that of the adjacent portions of the artery from which it arises; the artery reacts immediately after ventricular systole, and so causes a second wave to enter the sac, at a moment when, owing to the tardiness of its recoil, from defective elasticity, it is still in process of reaction. Hence, a collision between the volume of influx and that of efflux, and a sudden arrest to the contraction of the sac. If palpable tumor exist, the hand placed over it will perceive a uniform expansion, with an increase of tension coinciding with ventricular systole, which is immediately followed by a "movement of retraction,"† and an impulse of arrest. Pressure upon the artery on the distal side of the aneurism will increase the force of the systolic impulse, but it will abolish the diastolic, by maintaining a state of continuous tension of the sac. Gendrin considers the second impulse to be distinctive of aneurism; in this opinion I entirely concur.

The usual acoustic signs of aneurism of the arch consist in two sounds which coincide with the double impulse when such is present, and closely resemble the sounds of the heart. So close, indeed, is the resemblance, that Laennec regarded the sounds heard in an aneurism close to the heart, as being actually those of the heart itself, propagated to the aneurism. The frequent existence, however, of two sounds in portions of the aorta remote from the heart, renders this doctrine untenable. No less inaccurate is the unqualified statement of Hope, that the first sound of an intra-thoracic aneurism "is a murmur."‡ The existence of murmur in thoracic aneurism is the exception.

The sounds of aneurism are usually louder than those of the heart in the same person; a circumstance attributed by Bertin to the echo of a large cavity, as in pectoriloquy. This is espe-

\* *Revue Médicale*, 1844, tom. iii., p. 508.

† Gendrin, *loco citat.*

‡ *Opus citat.*, p. 442.

cially true of the second sound, which, as Dr. J. W. Begbie avers, is accentuated in thoracic aneurism and in simple dilatation of the aorta. In aneurism, too, the second sound frequently possesses a "booming" character.\* Dr. George W. Balfour holds a similar opinion in regard to the significance of an accentuated second sound in the aorta.† Both these writers believe that the phenomenon is due to the recoil of an increased volume of blood upon the valves. This subject has been discussed in a previous chapter.‡ I will here only repeat, that in my opinion, an intensified aortic second sound indicates atheroma and dilatation of the aorta with hypertrophy of the left ventricle. It may, therefore, exist in connexion with aneurism of the arch in which these conditions are supplied, quite irrespectively of its locality. The second sound in the aorta may be sharp and clear, without being intensified; it may, likewise, possess a "booming" quality. The former of these characteristics may be exhibited in aneurism; the latter is peculiar to it.

The aneurismal sounds have been attributed to various causes. Gendrin believed that they are due to the shock of impulse exclusively; he accordingly designated them as the first and second "*bruit de choc*," respectively. To Gendrin's theory of the second impulse and sound in aneurism Bellingham objects: (a) if it were true, a second impulse and a second sound should exist in the aneurism of minor arteries, but such is not the case; and (b) the adjacent portions of the vessel are assumed to be sound, whereas they are usually unsound.§ To these objections it may be answered, that the elasticity of the secondary arteries is much less than that of the aorta, and that the walls of the aorta in the vicinity of the aneurism are shown to be functionally competent by their not being aneurismal.

Bellingham attributed both the sounds of aneurism to the friction of the blood against the orifice of the sac; the first, to

\* *Edinburgh Medical Journal*, vol. viii., 1862-3. Dr. Stokes had, eight years previously, mentioned a "ringing" second sound as characteristic of thoracic aneurism (*Diseases of the Heart and Aorta*, 1854, p. 539). He was, therefore, the first to call attention to this sign.

† *Edinburgh Medical Journal*, January, 1874.

‡ *Vide* Chapter ii., p. 138.

§ *Dublin Medical Press*, June 7th, 1848.

last mentioned cause, namely, passive regurgitation of blood into the sac during ventricular diastole, under the influence of gravity, he likewise attributes the second impulse in thoracic aneurism. Against this latter doctrine several objections may be urged, which may be briefly stated as follows. It would necessarily imply the coexistence of the visible throbbing of "unfilled arteries" in the neck, and regurgitant murmur in the sac, as remarked by Dr. Stokes, phenomena rarely witnessed in connexion with thoracic aneurism. As observed by Dr. Lyons, lowering the head to the level of the body, the patient being recumbent, will not modify, much less suppress, the second impulse and sound, where both exist, in thoracic aneurism, nor, in femoral aneurism with a single impulse, will the elevation of the leg above the level of the trunk develop a second impulse, as proved experimentally by the same observer. Finally, it implies the absence of vascular reaction in sound arteries, and ignores the reaction of the arch of the aorta. A force by which the closure of the valves is effected and a second sound is produced, must be competent to prevent the gravitation of a column of blood from the arteries of the neck; hence, the second impulse and sound of aneurism could not, on Bellingham's theory, coexist with a second cardiac sound. But their concurrence is certainly the rule.†

Doctor Lyons, in a very able article, has propounded a theory of the second impulse in aneurism, the existence of which he would justly regard as proof that the aneurism was saccular and belonged to the "false" variety.‡ He agrees with Gendrin in attributing the second impulse of the sac in thoracic aneurism, to the influx of a second volume of blood under the reaction of the adjacent portions of the aorta. The absence of a second impulse in abdominal aneurism, and in aneurism of the extremities, he accounts for by assuming a fusion at a certain point of the aorta, probably at the commencement of the abdominal por-

\* *Ibid.*

† This subject has been already referred to at p. 844, in connexion with the signs of dilatation of the aorta.

‡ *Dublin Quarterly Journal of Medical Science*, vol. ix., 1850, p. 319.



arterial reaction; "that of the ventricular systole, being, as it were, overtaken by that of the arterial." To the want of concurrence of these two forces in saccular aneurism, the second impulse is, in his opinion, due; and to their fusion in aneurisms below the diaphragm, the absence of a second impulse is to be attributed. Against this theory, which would afford the most rational explanation that has been yet offered of the absence of a second impulse in abdominal aneurism, the objection may be urged that, carried to its legitimate conclusion, it would imply that the pulsation of abdominal aneurism, and the ordinary pulsation of distant arteries, could never precede the second sound of the heart, inasmuch as one of the assumed factors of such pulsation, the wave of aortic reaction, must necessarily be posterior in time to the second sound. It is, however, well known that both the phenomena referred to usually take precedence of the second sound.

I believe shock to be the chief factor in both the aneurismal sounds; whilst the density of the sac and surrounding structures accounts for their comparative intensity. I cannot pretend to explain the "booming" quality which occasionally distinguishes the second sound in aneurism, except on the questionable hypothesis of a momentary extrication of gas within the sac during ventricular diastole. On the cessation of systole, the sac becomes relaxed by reflux of the blood within it; into the virtual vacuum so produced, it is not inconceivable that gas may, for the moment, be evolved, to be again taken up by the blood under the increased pressure of the succeeding systole of the ventricle. Bernard has found that, during digestion, arterial blood contains 5 to 6 per cent. of free carbonic acid, which is readily evolved *in vacuo*. The proportion gradually decreases during the interval between meals, and after twenty-four hours' fasting, not a trace of carbonic acid is to be found in the arterial blood.\* It would be interesting to determine whether the "booming" sound of aneurism is in any degree influenced by digestion.

\* Quoted by Flint (*Physiology of Man*, p. 464, foot note), from unpublished lectures delivered in 1861.

systemic sound; whereas, in false aneurism of the large arteries, the sound is double. This doctrine is, however, inadmissible. True aneurism is rarely met with except in the ascending aorta, and here it yields two sounds, which are those of the heart, reinforced by the density of the arterial walls; whilst false aneurism not unfrequently yields a single and systolic sound only.

Murmur is of much less frequent occurrence in thoracic aneurism than impulse or sound. It is usually single, systolic, and blowing, but it may be "booming" in quality.\* The murmur of thoracic aneurism is, however, occasionally double, and still more rarely, a diastolic murmur only exists. Dr. Bellingham has published the details of 12 cases of thoracic aneurism, in all of which a double sound, normal or abnormal, was present; in most of these cases a double impulse likewise existed; in 2 instances a murmur accompanied or replaced the first sound: in one, murmur replaced both sounds, and in one, a murmur replaced the second sound. In 8 of these cases a double sound, closely resembling the sounds of the heart, existed.† With reference to this point, the following evidence is furnished by Table XVI. Impulse was observed in 29 cases; it was single and systolic in 22, and double in 7; it did not exist in 32 cases; and was not referred to in 7. The presence or absence of aneurismal murmur was noted in 61 cases; of these, it existed in 20; in 16 of this number it was single and systolic, and in one it was single and diastolic; it was double in 3 instances, and was absent in 41 cases.

The presence or absence of aneurismal sound, as distinct from murmur, was noted in 61 cases; it was present in 11 instances, in 2 of which it was single and systolic, and in 9 it was double. Sound was not present in 50 cases. A double impulse and a systolic sound coexisted with a diastolic murmur in a single instance. The existence of a diastolic murmur in aneurism is very rare; I have not met with an example of it. In one instance only I have noted a murmur of diastolic rhythm in *connexion* with aneurism of the ascending portion of the arch; but

\* *Vide* Case 69, p. 759.

† *Dublin Medical Press*, April 19th, May 24th, and June 7th, 1848; see also Dr. Greene's cases in *The Dublin Journal of Medical Science*, vols. vii. and x.

termination of the aorta. In the secondary instance (No. 15), in Table XVI., in which diastolic murmur existed, the aneurism was likewise connected with the ascending aorta. In another case (No. 60) a diastolic murmur was occasionally heard in connexion with an aneurism of the thoracic aorta. I must, therefore, regard diastolic murmur as an exceptional phenomenon in aneurism. It is, I believe, in most cases due to aortic regurgitation; but that it is occasionally intrinsic to aneurism must be admitted.

The murmurs of aneurism arise from the same causes as those of the heart, namely, friction and vibration. The doctrine of unequal pressure and velocity of current arising from abrupt constriction of a conduit, as first propounded by Corrigan, subsequently developed by the experiments of Chauveau, and by those of Dr. Leared,† may be studied with advantage in reference to this subject, which has been already discussed at page 181 and page 258.

As principal or contributory causes of aneurismal murmur, I may briefly advert to: (a) the figure, the relative diameter, and the actual state of the orifice and the walls of the sac; (b) the contents of the sac; and (c) the force of ventricular contraction. A relatively narrow orifice with a rough margin, a globular and flaccid sac springing from the vessel at an angle and containing fluid blood, and vigorous contraction of the left ventricle, constitute the most favourable conditions for the development of murmur.

Of these, the last mentioned is the principal factor of murmur; hence, at the approach of death, murmur, whether cardiac or aneurismal, becomes indistinct or inaudible, from failure of the heart. The absence of one or more of the other conditions mentioned may modify or exclude murmur, even though the heart act with energy.

Thoracic aneurism may be now considered in its clinical aspect, with a view to differential diagnosis. It may present, in a

\* Case 141.

† *Medical Times and Gazette*, October 31st, 1868. Dr. Leared has expounded his theory in an earlier publication, a reference to which I have not been able to find.



symptoms and signs, or afford evidence of so indefinite a character that it may be practically regarded as latent.

The existence in the chest of a centre of pulsation, distinct from that of the heart, would suggest aneurism, cancerous tumor, or pulsating empyema. The presence, in such centre, of a double sound, the second being louder than that of the heart, would, *pro tanto*, be in favour of aneurism, and tend to exclude cancer and empyema. A double impulse, the second being an impulse of arrest, would possess a similar but stronger significance. Single and systolic murmur would indicate aneurism and carcinoma in about equal proportion, and would exclude empyema. A double murmur, if associated with abnormal impulse in the situation of the ascending portion of the arch, would suggest aneurism rather than cancer or empyema.

The symptoms of excentric pressure, considered apart from other evidence, have no distinctive significance save in regard to their inconstancy or variation. If, in the progress of the case, symptoms of this character cease to be exhibited, or vary in degree, the presumption would be in favour of aneurism. The occurrence of paroxysmal dyspnoea, laryngeal or tracheal, associated with excitement of the circulation from physical or emotional causes, would likewise point to aneurism. But even in this respect, an abscess located between the trachea and oesophagus, may simulate aneurism. The following example came under my own notice.

A man, aged fifty, was admitted into the Mater Misericordiae Hospital on the 13th of April, 1864. He was in a state of imminent asphyxia, with cold extremities, lividity of surface, and a loud laryngeal stridor.\* The pulse was rapid, feeble, and intermittent, and deglutition, even of liquids, was attended with the greatest difficulty. The chest was universally resonant, and no evidence of pulmonary or vascular disease could be detected. Tracheotomy was performed on the evening of the 13th, and was followed by complete relief from all the urgent symptoms. On the 18th he had another paroxysm of dyspnoea which nearly proved fatal. On being raised in bed he coughed up about a

\* *Proceedings of the Pathological Society of Dublin*, vol. ii., part ii., p. 143.



chial rales were audible all over the chest. He could now swallow solid food. He had a third paroxysm on the 20th, and he died worn out on the 22nd. The viscera were healthy; a large abscess, which had been recently filled with pus, but was now empty, was found between the trachea and œsophagus. It extended from the larynx downwards into the posterior mediastinum, where it ended in a *cul de sac*. The arytenoid cartilages were necrosed; they were all but detached by ulceration from the cricoid, and had fallen forward upon the glottis. A large, ulcerated opening extended from the œsophagus into the abscess, whilst another and much smaller aperture connected the abscess with the trachea. Through the former opening, which had evidently been formed by a perforating ulcer of the œsophagus, particles of food had escaped and given rise to the abscess, whilst the paroxysms were the result of spasm from pressure upon the trachea, and latterly, of dislocation of the arytenoid cartilages.

An example of laryngeal distress and dysphonia, with immobility of the vocal chord, from paralysis of the right recurrent laryngeal nerve produced by the pressure of a malignant tumor of the œsophagus, has been published by Dr. Morell MacKenzie.\*

If, on the other hand, the signs of persistent excentric pressure within the chest coincide with cancerous tumor developed externally, the evidence, so far, would be in favour of intra-thoracic cancer.

Pulsating cancer is further distinguishable by the fact that the force and diffusion of impulse are not in proportion to the degree and extent of dulness; nor does the centre of pulsation correspond to that of dulness; and, further, the superficial thoracic veins on the side affected by intra-thoracic cancer are usually enlarged and varicose, whilst those related to an aneurism are never varicose, although they may be distended.† Pulsating emphysema, of which three remarkable examples have been published by Dr. Robert L. MacDonnell,‡ and one by Dr. Stokes,§

\* *Lancet*, May 30th, 1874.

† *Vide* Stokes in *Dublin Journal of Medical Science*, vol. xxi., p. 227. Also a case by Dr. O'Ferrall, *Proceedings of the Pathological Society*, April 23rd, 1842.

‡ *Ibid.*, vol. xxv., p. 1.

§ *Diseases of the Heart and Aorta*.

heart, which is displaced to the right side, by its being preceded by the symptoms and signs of acute inflammation on the left side of the chest, and by the presence of a single and systolic impulse communicated from the heart, and unattended with murmur, sound, or thrill. The foregoing evidence, positive and negative, will suffice to distinguish pulsating empyema from aneurism, even in the absence of metallic phenomena in the left side of the chest, which would be in themselves conclusive.

Laryngitis may be mistaken for thoracic aneurism, owing to the attendant symptoms of hoarseness or aphonia, dyspnoea, and stridor. The distinction would rest mainly upon the positive result of laryngoscopic inspection, and the absence of other symptoms, and of all the intrinsic signs of aneurism. The opposite and more serious error, that of mistaking aneurism for laryngitis, may be likewise committed. Dr. Gairdner mentions a case in which this mistake was actually made by an expert in laryngeal disease; and quite recently a similar case was published in which tracheotomy was actually performed with a fatal result. In this case the aneurism was latent, and the operation was demanded by the urgency of the symptoms.

Neuralgia or rheumatism engaging the muscles of the chest, owing to the attendant symptom of radiating or fixed pain, may be likewise confounded with aneurism; so also may tuberculosis and cirrhosis of the lungs, from the occurrence of hæmoptysis. But if a suspicion of aneurism be once aroused, the distinction from these diseases cannot be difficult. An error of diagnosis can arise only from the idea of aneurism *not* having been presented to the mind of the physician.

Doctor George W. Balfour has recently pointed out another possible source of error in the diagnosis of thoracic aneurism.\* A systolic murmur, with visible pulsation localized in the inner portion of the left second intercostal space, may arise from mitral reflux of anæmic or organic origin, propagated into the left auricular appendix, as first mentioned by Naunyn; or a presystolic murmur from mitral narrowing, similarly propagated,

\**Medical Times and Gazette*, December 12th, 1874.

in the pulmonary artery, may be likewise presented in the second interspace, but closer to the sternum, as first pointed out by Quincke. It was attributed by him to flattening of the pulmonary artery against the anterior wall of the chest during ventricular systole, the left lung having been permanently retracted from antecedent disease. Dr. Balfour details four cases in which these phenomena were exhibited. He observes, however, that the distinction from aneurism was readily made in all these cases by the absence of signs of excentric pressure, and by the more feeble impulse at the second centre of pulsation, as compared with that of the heart.

In an earlier publication on the same subject, Dr. Balfour had suggested a very ingenious means, by which, having reference to the relative rhythm of the cardiac and the abnormal pulsation, he could readily determine whether the latter was auricular or arterial. It consists of two pellets of bees' wax, with a bristle, supporting a small paper flag, fixed in each. One of these pellets is attached over the apex of the heart, and the other at the seat of abnormal pulsation. If the superior pulsation precede the inferior, as indicated by the movement of the flags, it would be regarded as auricular; if it accompany or succeed the inferior pulsation, it would indicate aneurism or pulsation in the pulmonary artery. The distinction between the two latter should be made by reference to the signs of excentric pressure, and relative force of pulsation, as already mentioned.\*

On Quincke's theory, murmur should always accompany visible pulsation of the pulmonary artery; but such is not the case. I would further remark, that the murmur of mitral stenosis is not usually audible in the left second interspace, although the attendant pulsation of the left auricular appendix is frequently visible there. But the presystolic rhythm of both these phenomena would, in any case, suffice to exclude aneurism.

The general symptoms and signs most distinctive of thoracic aneurism are: a pulsating tumor yielding a single impulse and a double sound, distinct from those of the heart, the second

\* *Edinburgh Medical Journal*, January, 1874.



second sound louder than that of the heart; paroxysmal or remittent pains of a radiating or shifting character; and the evidence of excentric pressure upon adjacent parts. None of these are, however, of constant occurrence, even in aneurisms having the same origin and relationship. Hence, the diagnosis must rest upon a rational basis, and be deduced from a careful consideration of *all* the features presented in each case.

The limits of variation are pretty well defined in regard to (a) true, (b) false, and (c) varicose aneurism, respectively; they are also, but less definitely, fixed in relation to the portion of the aorta whence the aneurism has arisen. It will be therefore necessary to review briefly the semeiology of thoracic aneurism under each of the foregoing heads.

True aneurism is rarely met with except in the ascending portion of the arch; it is tubular or fusiform, and cannot attain large dimensions without undergoing conversion into the false variety, by rupture of its internal tunics. Owing to this circumstance, and the usual absence of clot or deposited fibrin, true aneurism is characterized by few and indefinite signs. Diastolic impulse, murmur, or thrill, are seldom exhibited, because the necessary conditions for these phenomena, lateral deviation of current, and roughness of surface, are not present,\* and single and systolic bruit is not a sign of true aneurism, as stated by Bertin. Hence, the diagnosis of true aneurism must be in the highest degree doubtful. Indeed, the only positive sign which it may exhibit is an accentuated second sound, but this is equally characteristic of simple dilatation with atheromatous rigidity of the aorta.

The symptoms exhibited by a false aneurism within the chest, irrespectively of intrinsic pain, will depend upon the organs or parts implicated in its growth; these have been already considered in detail. I shall only add the following clinical remarks suggested by the cases herewith given.

\* Cruveiller holds that clotting cannot take place in an artery without previous erosion or inflammation of its internal surface; and F. Niemeyer (*Medical Times and Gazette*, January 22nd, 1870), declares that roughness of the *intima*, constituting *endarteritis deformans*, is an essential condition of fibrinous deposit in an artery.



ing nearly to occlusion, need not necessarily cause dyspnoea, laryngeal cough, stridor, or paroxysmal dyspnoea; that vocal vibration may exist in a lung from which air has been all but excluded by pressure upon the primary bronchus of that side, but that it is abolished by solidification of a lung so circumstanced; and finally, it shows that an aneurism may open simultaneously into the oesophagus and one of the bronchi.

Case 138, shows that saccular aneurism, with a large and smooth-edged orifice, an empty cavity, and flaccid walls, may present no intrinsic physical signs, and if small, deep-seated, and remote from the walls of the chest, may afford no evidence of its existence save that arising from excentric pressure; and that the most aggravated dyspnoea and stridor, from spasm of the larynx, may be due to reflex irritation of the recurrent laryngeal nerves. It also proves that feeble respiration in one lung, accompanied by laryngeal spasm, may be caused by pressure upon the trachea, and that, in such cases, laryngotomy may be followed by complete relief from urgent symptoms.

With regard to relative frequency of occurrence, in combination and singly, the various tactile and acoustic phenomena of thoracic aneurism, as deduced from the 17 cases (comprising 18 aneurisms\*) which I have personally noted, might be set down in the following order:

(a) Double impulse and double sound, without murmur	...	3 cases.
(b) Systolic impulse and double sound, without murmur	...	4 "
(c) Systolic impulse and systolic murmur, without sound	...	3 "
(d) Double impulse and double sound, with systolic murmur	...	1 "
(e) Systolic impulse and double murmur, without sound	...	1 "
(f) Double murmur, without impulse or sound	...	1 "
(g) No impulse, sound, or murmur	...	5 "
		—18

It will thus be seen that:

Double sound existed in	...	8 cases.
Single and systolic impulse in	...	8 "
Double impulse in	...	4 "
Single and systolic murmur in	...	4 "
Double murmur in	...	2 "

and that, in 5 out of the 17 cases, the aneurism was latent, *quoad* tactile and acoustic signs.

\* In one case (No. 150) there were two aneurisms.

that variety of the disease in which blood passes from one portion of the vascular system to another." Aneurism within the chest may establish communication with (a) the chambers of the heart, (b) the pulmonary artery, or its branches, and (c) the descending cava, or the innominate veins.

According to Peacock,\* the first recorded example of varicose aneurism was one opening into the left auricle, and published by Beauchene in 1811. Wells, in 1812, published a case in which an aneurism of the aorta formed a communication with the pulmonary artery. In 1832 an example of varicose aneurism implicating the superior vena cava, was recorded in *The Lancet*.

Cases were subsequently recorded by Curling (1838), Munro, (1839), Reid (4 cases), and Thurnam (10 cases, including the preceding, 1840). Peacock gives reference to 33 cases, not including 6 referred to by Thurnam, as contained in various museums, and 4 met with by Rokitansky.

In 18 of this number, the aneurism arose from one or more of the sinuses of Valsalva or from the aorta immediately above the sinuses; in 12, from the ascending portion of the arch, above the points mentioned; in 2, the aneurism engaged the entire arch except the termination of its descending portion; and in one case there were two aneurisms, one in the ascending, and another in the descending portion of the arch, the latter opening into the left branch of the pulmonary artery.

Only two examples of varicose aneurism have come under my notice. In one of these, the aneurism opened into the right ventricle (Case 69, p. 759), and in the other (Case 141), into the superior cava. Two additional examples have been recently recorded in *The Proceedings of the Pathological Society of Dublin*; one by Dr. Gordon, in which an aneurism of the left anterior sinus of the aorta opened into the left ventricle of the heart,† and another, by Dr. Bennett, in which an aneurism of the right sinus communicated also with the left ventricle.‡

Doctor McDowel exhibited before the Pathological Society, in January, 1849, an example of varicose aneurism springing from the aorta, half an inch above the valves, and opening into

\* *Transactions of the Pathological Society of London*, vol. xix., p. 111.

† Vol. ii., part i., new series.

‡ Vol. iii., part ii.

subclavicular region, and yielded a systemic impulse, accompanied by fremitus, and a harsh systolic murmur audible only between the middle line and the right nipple.

Hope, in 1839, published a remarkable case of aneurism of the aorta immediately above the valves, communicating with the right ventricle by two apertures. The symptoms and signs, which followed a strain in lifting a weight, and a "creak in the heart," accompanied by faintness, were lividity, œdema, and a jerking pulse, purring tremor, double murmur, and a continuous rumble to the left of the sternum, from the second to the fourth rib.\*

In the case which came under my own notice, through the favour of Dr. Stokes, an aneurism of the size of a tennis ball, arising from one of the aortic sinuses, communicated with the right ventricle by an aperture of the size and figure of a shirt button-hole. The symptoms were, general venous congestion with œdema; and the physical signs, *frémissement*, with a loud and booming double murmur at midsternum, audible to the patient, and compared by him to "a woman churning;" all which signs were subsequently presented with greatest intensity at the apex of the heart, but diffused over the entire left front of the chest. The diagnosis of aneurism opening into one of the ventricles was presumptively made.†

Thurnam has included in his list of cases of varicose aneurism three examples of aneurism of the aorta opening into the heart. In two of these cases, observed by Curling and Reid respectively, a communication was established with the right auricle; and in the third, supplied by himself, with the right ventricle by two apertures. In this case, a loud superficial and continuous sawing murmur, accompanied with purring tremor, was audible in the left second intercostal space, an inch and a-half from the sternum, but loudest over a space not larger than a shilling. The heart had been displaced to the left by effusion into the right pleural cavity.‡

Aneurisms projecting into the chambers of the heart, but not communicating with them, must be distinguished from those

\* *Diseases of the Heart and Great Vessels*, third edition, p. 466.

† *Vide* Case 69, p. 759.

‡ *Medico-Chirurgical Transactions*, vol. xxiii., p. 323.



and signs must obviously differ in the two classes of cases; hence the necessity for a clinical distinction between them.

Professor Smith's case was one of the earliest recorded examples of this kind. A man, aged thirty, was admitted into hospital suffering from urgent dyspnœa, which was accompanied by lividity, cedema, and suppression of pulse. There was great extension of precordial dulness, with feeble action of the heart. He died on the following day. An aneurism, with an orifice as large as a shilling, arose from the aorta one inch above the valves, and extended downwards behind the pulmonary artery, which lay across it, and gave the appearance of two tumors, each as large as a hen-egg. One of these projected into the base of the right ventricle near the pulmonary orifice, and the other into the superior and anterior portion of the left ventricle. The aneurism did not communicate with either ventricle.\*

An example of aneurism of one of the sinuses of Valsalva, projecting into the right auricle, was presented to the Pathological Society of Dublin by Dr. Jennings, in December, 1866.

Doctor Law exhibited before the same Society in February, 1870, an example of aneurism springing from the posterior sinus, advancing downwards and to the right, displacing the liver and causing slight jaundice. It projected into the right auricle of the heart, occluding the orifice of the inferior cava, and partially closing that of the superior. The most prominent symptoms were general venous congestion and dropsy.

Doctor Hanna has published a case in which an aneurism, as large as an orange, arose from one of the sinuses of Valsalva by an orifice two lines in diameter, and was imbedded in the wall of the left ventricle. The muscular substance had been entirely absorbed around the sac, which was formed by the thickened membranes of the heart, and contained laminated fibrin. The patient was a man, aged thirty-one, who had sustained a severe fall from a horse, and shortly afterwards complained of severe pain in his chest, palpitation, and dyspnœa; he also spat blood. The symptoms were orthopnœa, and feeble but regular pulse. In the left second interspace there was a double impulse accom-

\* *Dublin Journal of Medical and Chemical Science*, vol. ix. See also a case by Professor Harrison, *ibid.* vol. xv.



throughout the precordium, but were most distinct in the second intercostal space.\*

Varicose aneurism between the aorta and the pulmonary artery is characterized by symptoms and signs nearly identical with that variety of the disease examined in the preceding paragraphs. It may therefore be discussed here. Dr. Thurnam has collected from various sources eleven examples of this variety of aneurism, and of five of these he has given a full history.†

A case is given in detail by Professor Smith, which may be briefly referred to. A man, aged twenty-two, had been subject to vertigo with temporary loss of vision, dyspnoea, faintness, and chills; then followed general venous congestion, cough, orthopnoea, anasarca, and intermitting pulse. He was troubled by great anxiety and disturbing dreams. There was extension of precordial dulness, with tumultuous action of the heart, *frémissement* which was perceptible through the bed-clothes, and systolic murmur. The man felt "as if there was a living bird in his chest." He subsequently had epileptic seizures, in one of which he died. The lungs were congested, and the aorta communicated with the pulmonary artery, near its origin, by an opening with thick and rounded edges.‡

Doctor Peacock has published a case in which an aneurism of the ascending aorta opened into the pulmonary artery by an orifice two lines by four in diameter; it projected also into the right ventricle, destroying one of the pulmonic valves. The heart was enlarged, and the aortic valves were disorganized. The symptoms were dyspnoea, general lividity, and oedema of the feet. There was extension of precordial dulness, with systolic murmur all over the precordium, and postdiastolic murmur at the right base. The second sound was very sharp in the left second interspace near the sternum. This latter phenomenon he attributed to the tension of the sac during diastole.§ In a case by Dr. W. Wade, the diagnosis was made from the existence of a basic diastolic murmur, not due to inadequacy of the aortic valves.||

\* *Ibid.*, March 1st, 1835. † *Loco citat.*

‡ *Dublin Journal of Medical Science*, vol. xviii. See also the case given by Hope, *opus citat.*, p. 469.

§ *Transactions of Pathological Society of London*, vol. xix., p. 111.

|| *Medico-Chirurgical Transactions*, vol. xlv.

communicating with it. The two classes of cases are characterized by special clinical features, and should not be confounded.

An example of compression of the pulmonary artery by an aneurism of the ascending aorta has been published by Sir D. Corrigan. In addition to the ordinary signs of aneurism, there was general venous congestion, with dropsy and delirium.\*

Of aneurism, communicating with either vena cava, several examples are on record. One of the earliest was that published by Mr. Syme, in which an aneurism of the size of an orange, arising from the abdominal aorta above its bifurcation, opened into the inferior cava by an aperture as large as a sixpenny piece. There was cedema of the lower half of the body; also a loud whirring noise audible to the patient and his friends.†

Doctor Thurnam has published‡ a case of a large aneurism of the ascending aorta, opening into the superior cava by an orifice a quarter of an inch in diameter. There was cyanosis, with cedema of the upper half of the body, and a loud buzzing noise over the vena cava. This case was observed by Dr. Thurnam in 1833. The next, and the most important case of this kind, because a positive diagnosis of the lesion was made from the phenomenon exhibited, has been recorded by Dr. Mayne.§ A large aneurism, engaging the entire arch of the aorta, had compressed and occluded both innominate veins and the superior cava, with the latter of which vessels it had formed a communication by an opening of the size and figure of the button-hole of a shirt, crossed by a delicate band. The patient, a woman aged fifty, whilst washing in a stooping posture, was suddenly seized with symptoms of strangulation, accompanied by dizziness. Her face became livid; her breathing embarrassed. When the patient was seen by Dr. Mayne, the upper half of the body was puffed and deeply cyanosed, the eyes protruded, the veins of the neck

\* *Lancet*, February 7th, 1829. See also a similar case by Mr. Carmichael, *Proceedings of Pathological Society of Dublin*, December 5th, 1840.

† *Edinburgh Medical and Surgical Journal*, vol. xxxvi., 1831, quoted by Thurnam (*loco citat.*), who also refers to two other similar cases, one by Mr. Robinson, of Camberwell, and another by Dr. John Reid; in neither of which, however, was a stethoscopic examination made.

‡ *Loco citat.*

§ *Dublin Quarterly Journal of Medical Science*, November, 1853.

the lower half of the trunk and the lower extremities, were quite free from oedema, and of their natural colour, were in no degree congested. The pulse was jerking. The physical signs were a heaving systolic impulse accompanied by fremitus, and a whirring systolic murmur, immediately to the right of the sternum at the level of the second rib. This murmur was diffused all over the front of the chest; but its point of greatest intensity corresponded to the junction of the second costal cartilage with the sternum, whilst the fremitus was transmitted into the great veins of the neck. The heart was perfectly healthy.

In the case (141) which came under my own observation, there was great venous turgescence, with deep cyanosis and depressed temperature of the upper half of the body, systolic impulse, thrill, and murmur in the seat of the aneurism; the latter transmitted into the carotids. A diastolic murmur likewise existed at mid-sternum, but it was not audible at the seat of impulse and systolic murmur. There was no visible pulsation or thrill of the cervical arteries or veins.

*The symptoms and physical signs* exhibited in the foregoing cases resolve themselves into two groups; namely, 1st, those which are characteristic of an aneurism communicating with (*a*) one or more of the chambers of the heart, (*b*) the pulmonary artery or one of its branches, (*c*) one of the venæ cavæ; and, 2nd, those which arise from the intrusion, without rupture, of an aneurism into the chambers of the heart, or into one of the vessels just named. The cases in each of these groups are marked by special features sufficiently distinctive of their nature:

The irruption of the contents of an aneurism into the heart, or one of the great vessels named, is usually associated with a definite strain or shock. It is attended with a feeling of suffocation and faintness, and is immediately followed by great dyspnoea, feeble and fluttering pulse, venous congestion more or less general according to the point of abnormal influx, with coextensive anasarca and depression of temperature. The physical signs developed are, thrill associated with impulse, and a harsh, prolonged, systolic murmur of a buzzing character at the seat of aneurism, and transmitted to a variable distance in the line of



the two murmurs, owing to the presence of an intervening rumble, not unfrequently seem to be continuous. The existence of arterial throbbing in the neck, and a jerking pulse at the wrists, depends upon reflux into a large aneurismal reservoir during diastole; it therefore may, or may not, according to the size and capacity of the sac, characterize an arterio-cardiac or an arterio-venous aneurism. The phenomena, then, which distinguish varicose aneurism, viewed in the abstract, are three in number; namely, extreme venous congestion suddenly developed; thrill; and a buzzing murmur of systolic rhythm transmitted in the line of abnormal circulation.

Aneurism communicating with the heart is not characterized by any symptoms or signs by which it may be distinguished from other forms of varicose aneurism within the chest. A sudden transfer of the seat of murmur and thrill, from the base to the apex of the heart, as occurred in Case 69, might be regarded as proof of the irruption of an aneurism into either ventricle.

Aneurism opening into the pulmonary artery is especially characterized by sudden and most urgent dyspnoea, unaccompanied by spasm or stridor, with bloody expectoration. Where aneurism of the ascending portion of the arch of the aorta had been previously verified, the sudden occurrence of these symptoms might be regarded as pathognomonic of a communication between it and the pulmonary artery. Death, in such a case, may occur very rapidly by asphyxia.

The symptoms which distinguish arterio-venous aneurism are those which result from the sudden introduction of retrograde arterial pressure into a definite portion of the venous system. The veins, distal and tributary to that with which the aneurism has established a communication, are engorged, and the corresponding portions of the body are cyanosed and depressed in temperature, whilst all other portions of it retain their normal colour and heat. Hence, a varicose aneurism of the abdominal aorta and the inferior cava is distinguished by venous congestion, cyanosis, and oedema of the lower portion of the



of the aorta and the superior cava, by a similar condition of the upper half of the trunk, the upper extremities, and the head and neck, as illustrated in the cases published by Thurnam, Mayne, and myself.\*

Aneurisms communicating with the heart, the pulmonary artery, or the superior cava, have, without an exception known to me, taken origin from the ascending portion of the arch of the aorta.

The simple projection of an aneurism of the aorta into one or more of the chambers of the heart, is attended only with symptoms of obstructed circulation proportionate to its interference with the passage of the blood, and by acoustic signs having reference to its effect upon the orifices and valves.

The modification of the symptoms and signs of thoracic aneurism, dependent upon its position in the chest, may be now considered. The posterior wall of the chest is rarely affected by an aneurism of the first portions of the arch of the aorta. Not unfrequently, however, a large aneurism of the upper portion of the ascending aorta approximates the posterior thoracic wall, and is perceptible in the right scapular region by modified impulse, dulness, and sound. It is still more frequently discoverable in this situation by the respiratory and vocal phenomena arising from compression of the lung; namely, bronchial or tracheal respiration, bronchophony, and vocal fremitus.†

Aneurisms of the descending portion of the arch, and those of the descending thoracic aorta, directly implicate the posterior wall of the chest on the left side in a very large percentage of cases. Indeed, aneurism of the last mentioned portion of the vessel rarely fails to penetrate the posterior wall on that side, and to present a visible tumor in the scapular or infra-scapular region. Erosion of the vertebræ and the ribs are amongst the ordinary consequences of aneurism of these portions of the aorta. Hence, the two kinds of pain, paroxysmal or intermittent, and fixed or boring, already mentioned. The boring pain of vertebral erosion is always associated with local tenderness to per-

\* See pp. 1126 and 1127.

† Vide Case 142.

destruction of the vertebræ has been effected;\* but most certainly its absence is in the highest degree exceptional under such circumstances.

The existence of a double sound over a limited portion of the vertebral column, especially if associated with pain and tenderness in the same situation, is, in the highest degree, suggestive of aneurism; because, as well observed by Laennec and Bertin, the two sounds of the heart are rarely audible in the back. A perceptible impulse on either side of the dorsal spine is likewise suggestive of aneurism; and, owing to the normal position of the thoracic aorta to the left of the vertebral column, such impulse would possess more significance if felt upon the right side, as remarked by Dr. Greene. Darting or shooting pain in the line of the ribs, is a common result of pressure upon the intercostal nerves.

Displacement of the lungs, the heart, or the liver, may result from the growth of an aneurism within the chest. The left lung may be displaced upwards, and condensed, and local pleuritis with friction sound, produced by the pressure. The heart may be likewise dislocated upwards,† forwards,‡ or backwards. Displacement of the heart forwards is characterized by the "double jogging" impulse of Hope; and, in the case mentioned by Bellingham just referred to, it was associated with diastolic murmur which ceased after the retrocession of the heart. In the case recorded by Dr. Mayne, the cardiac impulse was violent, and its area was extended. In a case published by Dr. Douglas Powell, a small aneurism arising from the aorta, one inch above the valves, had descended in front of the heart, displacing the apex backwards.§

Amongst the symptoms especially suggestive of aneurism is a harsh and loud cough of metallic resonance. Even in the absence of stridor, dysphonia, and of any of the physical signs of

\* See a case by M. Valleix, *Archives Générales de Médecine*, vol. xxii., 1850.

† *Vide* Case 137.

‡ *Vide* a case by Hope, *opus citat.*, p. 447; also one by Mayne, *Proceedings of Pathological Society of Dublin*, June 24th, 1873; and another by Bellingham (case 12), *loco citat.*

§ *British Medical Journal*, March 6th, 1875.

in the chest, has led to the diagnosis of aneurism which was subsequently verified by dissection. The aneurism was found to have arisen from the transverse portion of the arch, and pressed upon the left bronchus, into which it ultimately opened.\*

Doctor Stokes mentions a case seen by him in consultation with the late Sir P. Crampton, in which the only symptom produced by a large aneurism of the ascending and transverse portions of the arch of the aorta, was a single sonorous inspiration, like that of whooping cough, at the termination of a fit of coughing.†

Recurrent hæmoptysis, in the absence of laryngeal, pulmonary, and cardiac disease, is likewise eminently suggestive of thoracic aneurism. It may occur by the "leakage" of an aneurism into the air passages, or it may be the result of congestion from the pressure of an aneurism upon the lung-structure, or the pulmonary veins; or from the irruption of an aneurism into the pulmonary artery. When hæmoptysis is produced by the direct communication of an aneurism with the bronchial system, the sputa are either streaked with, or entirely composed of, arterial blood. Pulmonary apoplexy, by aspiration of blood into the air passages, and evinced by the signs of solidification more or less extensive, may complicate an accident of this kind, or it may arise from pressure and local congestion, followed by rupture of the distended vessels. In the latter event, passive pneumonia, attended with the usual physical signs, will follow unless death occur within a short period. The sputa from pulmonary congestion exhibit a uniform intermixture of blood.

Doctor Gairdner holds that hæmoptysis, accompanied by laryngeal stridor not due to ulceration of the larynx, and in the absence of pulmonary disease or solidification, can arise only from a leaking aneurism.‡ This proposition cannot be disputed. In such cases, however, other symptoms and certain signs of aneurism, are usually exhibited, especially laryngeal cough and inequality of respiration.

\* O'Ferrall, *Proceedings of Pathological Society of Dublin*, March 24th, 1860.

† *Opus citat.*, p. 559.

‡ *Clinical Medicine*.



the subclavian arteries, accompanied by tremor and acoustic murmur, as indicative of aneurismal dilatation of the superior wall of the transverse portion of the arch. The inflection of the subclavian arteries, whence the phenomena in question arise, is due, he thinks, to loss of elasticity and consequent elevation of the superior wall of the aorta.\* I have met with an example of this kind in the person of a female of middle age and a highly nervous temperament. The carotid and subclavian arteries were remarkably tortuous, and under the slightest emotional excitement they pulsated with great violence. Immediately above the sternal end of the right clavicle, the common carotid was sharply bent upon itself, and formed a visible tumor which yielded a throbbing pulsation closely simulating that of innominate aneurism.

The existence of an aneurism of the arch of the aorta may be entirely masked, in regard both to symptoms and signs. Such examples are designated *latent*. In exceptional instances, large tubular aneurisms involving the entire arch may be unattended by symptoms or signs of any kind, except neuralgic pains diffused over the chest, shoulders, and neck, and an accentuated second sound in the aorta.† Small aneurisms arising from the left extremity of the arch, and involved in the substance of the left lung, are, however, those which are most frequently latent. The existence of such has been, in many cases, announced only by the occurrence of a fatal hæmorrhage. Several examples of this kind have been exhibited before the Pathological Society of Dublin by Drs. Mayne, O'Ferrall, Lees, Hudson, Banks, and Symes.‡

With regard to the use of the sphygmograph in the diagnosis of thoracic aneurism, Mr. Mahomed declares that, "If the aneurism is one of an artery going to the upper extremity, and the aorta be not involved, valuable evidence will be afforded:

"If the aneurism is one of a similar artery, involving the aorta, frequently no difference can be detected in the pulsation of the radials.

\* *Archives Générales de Médecine*, in *London Medical Record*, June 10th, 1874.

† *Vide* Case 139.

‡ *Vide Proceedings, passim*.



ascending part of the arch, no difference in the pulse on either side can be detected.

"If, of the transverse part of the arch, the pulse may or may not give evidence of its existence."\*

*Aneurism of the arteria innominata* may be now briefly considered, chiefly in reference to the differential diagnosis from aneurism of the aorta.

This subject has been treated exhaustively by Dr. Holland,† who has collected and epitomised from various sources 46 recorded examples of the disease. Innominate, as contrasted with aortic aneurism, presents the following distinctive peculiarities, as deduced by Dr. Holland from an analysis of these 46 cases. (a) More frequent appearance of external tumor; (b) more frequent dislocation of the larynx and trachea; (c) more frequent congestion of the right side; (d) less frequent occurrence of diffused or radiating pain in the chest; (e) more frequent occurrence of pain in the right side of the head and neck, and the right arm; (f) more frequent occurrence of cedema and paralysis confined to the right arm; (g) more frequent occurrence of diminished pulsation in the arteries of the right side of the neck and right arm; and (h) the decided effect of pressure upon the right carotid and subclavian artery in diminishing and arresting the pulsation of the sac.

Amongst the distinctive peculiarities of innominate aneurism, the seat of pulsation and sound behind the right sterno-clavicular articulation, the early appearance of a tumor above the clavicle, and of the signs of vascular obstruction on the right side, and the frequent displacement of the trachea and larynx to the left, deserve special notice. It may be further observed that, whilst the intrinsic signs are identical with those of aneurism of the arch, the signs of pressure upon the right bronchus and lung are less frequently exhibited than in the last mentioned disease.

Diminished force of circulation in the right carotid and subclavian arteries at an early period of the disease, is the most constant symptom of innominate aneurism. Dr. Sibson has

\* *Medical Times and Gazette*, August 30th, 1873.

† *Dublin Quarterly Journal of Medical Science*, vol. xiii., 1852.

the latter in nine-tenths of the cases. The aorta is frequently involved in aneurism of the arteria innominata. Towards the determination of this point, which is of the utmost importance in regard to surgical treatment, but in many cases attended with the greatest difficulty, the sphygmograph is capable of affording invaluable assistance. According to Mahomed, the exhibition of decided aneurismal characters in the pulse-tracing of the right side, might be deemed conclusive as *against* the supposition that the aorta was involved.

Doctor Stokes adverts to the singular variation of the voice, "from the deepest bass to a shrill treble," exhibited in a case of innominate aneurism which came under his notice.†

In the progress of growth, an aneurism of the innominate artery may transgress the middle line, and invade the parts on the left side of the neck, as occurred in a case reported by the late Dr. Hutton.‡ In a case published by Dr. McDowel, the greater portion of the tumor lay within the chest, where the physical evidence of its presence was masked by the intervention of the right lung between the tumor and the anterior wall of the chest.§ In this case, and in Dr. Hutton's, both of which were diagnosed at an early period, the first symptom exhibited was severe pain in the right side of the neck, the right shoulder, and ear. Recurrent laryngeal spasm, accompanied by paroxysms of dyspnoea from tension of the right recurrent nerve, may likewise result from the growth of an aneurism of the innominata. An example of this kind has been published by Dr. Hughes Bennett,|| and another by Dr. Gairdner.¶

The immediate *cause of death* and the mode of its occurrence in thoracic aneurism, are various. Death may be caused by intercurrent disease, acute or chronic, or by disease directly or remotely consecutive to the aneurism; as bronchitis from local

\* Croonian Lectures, *Lancet*, April 9th, 1870.

† *Dublin Journal of Medical Science*, vol. v.

‡ *Ibid.*, vol. xxv.

§ *Proceedings of the Pathological Society of Dublin*, vol. iv., part i.

|| *The Principles and Practice of Medicine*, second edition, p. 563.

¶ *Clinical Medicine*, p. 470.

phic softening or abscess of the brain, or by phthisis. An intimate association between aneurism and pulmonary tuberculosis has been alleged by Dr. Stokes, as a legitimate inference from clinical observation. I have met with one case (No. 142) which appeared to afford very decided evidence in support of this doctrine. The patient, a man aged fifty-four, with no hereditary predisposition, and no constitutional tendency to phthisis, died of aneurism of the ascending and transverse portions of the arch of the aorta, after an illness of thirteen weeks' duration, in the course of which no hectic symptoms were exhibited. Both lungs were studded with recent miliary tubercle. Several cases in which aneurism and pulmonary tubercle coexisted have been published in the *Proceedings of the Pathological Society of Dublin*. Rokitansky insists on a positive antagonism between aneurismal and the tuberculous diathesis.\* Dr. Walshe has met with only 2 examples of pulmonary tuberculosis in 16 cases.† The doctrine of affinity between aneurism and tubercle must, therefore, though probable, be regarded as still unproven.

With regard to the mode in which death has occurred, directly from thoracic aneurism, the 68 cases given in Table XVI. yield the following results: Death occurred by exhaustion from pain and insomnia in 11 instances; from repeated small hæmorrhages in 1; asphyxia 9; asthenia the result of mal-nutrition 5; syncope 5; coma 4; bronchitis 4; pleuro-pneumonia 1; and from rupture of the aneurism in 26 instances. Rupture into the pericardium occurred in 10 cases; into the trachea in 4; right pleura 3; left lung and pleura 3; left bronchus 2; left lung 1; left pleura 1; left bronchus and cesophagus 1; and externally 1. Rupture into the left lung or pleura was more frequent than into the right in the proportion of 8 : 3. As judged by the preceding results, then, death from thoracic aneurism is most frequently due to rupture of the sac. It results from other causes in the following order of frequency: exhaustion or debility, acute pulmonary inflammation, and coma.

Hæmorrhage from rupture of a thoracic aneurism may be

\* *Pathological Anatomy*, vol. i., p. 315.

† *Diseases of the Heart*, fourth edition, p. 499.



from repeated small bleedings.

Fatal syncope by loss of blood may result from a single and copious hæmorrhage, externally, into either pleura, the œsophagus, or the stomach; or by compression of the heart from the escape of a few ounces of blood into the pericardium.\*

The opening of an aneurism into the trachea or any portion of the bronchial system may be followed by death from asphyxia within a few minutes. The aperture may, however, be plugged by fibrin, and death postponed for an indefinite period.†

Hæmorrhage, by rupture of an aneurism upon the cutaneous surface, is necessarily preceded by visible tumor and slough, and is, therefore, never unexpected. On the detachment of the slough the first gush from the sac may be fatal, but this is rarely the case. It usually happens that, after the loss of a few ounces of blood, syncope supervenes, and hæmorrhage is arrested owing to the temporary debility of the heart and the formation of a plug of fibrin in the aperture. This object may be likewise effected by mechanical means.

Doctor Stokes mentions a case under the care of the late Dr. Osborne, in which an external opening by detachment of slough in an aneurism of the chest, was closed at each systole by a plug of fibrin; the plug receded with each diastole. It was at length dislodged, and a formidable gush of blood followed, but further hæmorrhage was stayed by the nurse, who plugged the opening with a cotton apron. By means of this *tampon* the patient's life was prolonged for several days.‡ In the course of the session 1864-5, Dr. Minchin reported to the Pathological Society a somewhat similar case.§ A large aneurism of the transverse portion of the arch of the aorta had pointed in the neck, and sloughed at its most prominent point. A slight oozing of blood occurred by filtration through the sloughing integument; this

\* Both Laennec and Hope declared they had never met with an example of rupture of an aneurism into the pericardium.

† Dr. Gairdner has recorded a case in which an opening into the trachea became blocked by a mass of fibrin, and death did not occur till four years afterwards. Mr. Liston lived five months after an opening into the trachea had been formed.

‡ *Opus citat.*, p. 580.

§ Vol. ii., part iii., p. 192, No. 29, Table XVI.



as a sixpenny piece was formed by detachment of a slough. This was, however, plugged by a mass of fibrin, which accurately closed the opening during the systole of the ventricle, but permitted a slight escape of blood during diastole. On the tenth day the opening had attained the size of a half-crown. In this opening a large globular mass of fibrin, which accurately fitted it, was seen to move alternately forwards and backwards with the systole and diastole of the ventricles. On the eleventh day from the first occurrence of hæmorrhage, this plug, which was as large as a man's fist, was suddenly shot out of the opening, followed by a gush of blood proving instantly fatal.

A still more remarkable example of protracted external hæmorrhage from an aneurism of the aorta, is mentioned by Dr. Stokes.\* The patient, a man aged fifty-six, under the care of the late Dr. Neligan in the Jervis-street Hospital, presented a large aneurism in the right mammary region. It had passed through the chest-wall, and it yielded a strong impulse and a double murmur; the voice was husky, and there was paroxysmal cough with dyspnœa. This tumor had existed for a year, and continued, during that period, to discharge blood in small quantity through a sinuous opening on its outer surface. Repeated hæmorrhage, in jets, occurred under Dr. Neligan's observation, but was easily restrained by mechanical means. Under a nutritious diet the tumor became solid, the pulsation diminished, cough and dyspnœa ceased, and the sinus entirely closed. The patient ultimately left hospital, declaring he felt quite well.

A case observed by the late Professor Smith is mentioned by the same writer, in which an external hæmorrhage from an aneurism of the aorta that had perforated the sternum, was restrained for ten days by a compress and roller. The patient ultimately died of exhaustion from continued leakage through the opening.†

A case, in which external hæmorrhage from an ulcerated opening in a large aneurism of the arch of the aorta was restrained for sixteen days by a plug of fibrin, has been published by Dr. W. H. Webb from the clinique of Dr. Da Costa. Dr. Webb gives

\* *Opus citat.*, p. 582.

† *Opus citat.*, p. 581.

which external hæmorrhage by rupture of the sac had occurred. The duration of life after the first hæmorrhage varied in these cases from seven weeks to a few seconds.\*

Serous membranes usually give way suddenly, and by rent, under the tension of an aneurism.

Hæmorrhage by rupture into either pleural cavity is usually fatal on the instant. Such, however, is not necessarily the case. As on the external surface of the body, hæmorrhage into a serous cavity may take place "by successive gushes, between the periods of which complete reaction may occur," as truly remarked by Dr. Stokes.

Laennec maintains that the left pleura is, more frequently than the right, the seat of hæmorrhage from rupture of a thoracic aneurism. In this opinion, which is borne out by the statistics furnished in Table XVI., I entirely concur.

Thurnam avers that the occurrence of a consecutive false aneurism of the intra-pericardial portion of the aorta is not possible, owing to the absence of an external or areolar coat in this portion of the vessel.† Hence, aneurisms within the pericardium usually prove fatal at an early period of their growth, and by simultaneous rupture of the sac and its serous investment. But the frequent occurrence of dissecting aneurism in this situation proves that the rule in question admits of many exceptions. Even the actual occurrence of hæmorrhage into the pericardium is not necessarily fatal on the instant. In the case of the late Dr. Ball, reported by Dr. Stokes, the patient survived, for one hundred and eight hours, the rupture of an aneurism into the pericardium by two small rents in the serous membrane.‡ In a case recorded by Dr. Finny, hæmorrhage into the pericardium, by rupture of an aneurism of the aorta immediately above the valves, was restrained by adhesion of the pericardium to the heart. Death did not occur till twenty-four hours after rupture took place, as indicated by the symptoms.§

\* *American Journal of the Medical Sciences*, No. 136, new series, October, 1874.

† *Medico-Chirurgical Transactions*, vol. xxi., p. 232.

‡ *Proceedings of the Pathological Society of Dublin*, April 25th, 1857.

§ *Ibid.*, vol. iii., part ii., new series; see also a case by Dr. Law, *Dublin Journal of Medical Science*, vol. xxi.

subcutaneous rupture of the sac, constituting secondary false aneurism, is seldom fatal for many days after its occurrence. The intrinsic signs are, however, in a great degree modified by such an event. A case has been recorded by Dr. Bellingham in which a thoracic aneurism, pointing externally, and characterized by a double impulse and a double sound, became diffused under the pectoral muscles. Of the previous signs, systolic impulse alone remained, but it was now accompanied by murmur.\*

Death from thoracic aneurism may occur by cerebral congestion and coma, as when the sac opens into the superior vena cava, or occludes it by compression; by spinal paralysis consequent upon rupture of the sac into the spinal canal; by sudden thrombosis of the aorta in connexion with a similar condition of the left ventricle;† or slowly, by inanition from compression of the œsophagus.

The *treatment* of thoracic aneurism should have reference to alleviation, and to cure. The paroxysmal and neuralgic pain of aneurism is promptly mitigated or suspended by the hypodermic use of morphia. One-sixth to one-fourth of a grain of the acetate or hydrochlorate of morphia, in aqueous solution, injected under the skin of the arm by means of the ordinary hypodermic syringe, rarely fails to bring, within a few seconds, partial or complete relief from pain, which lasts from two to six hours.‡ Opium, given in pill or in draught, is less efficacious. If the pain proceed from tension of the sac, as indicated by a feeling of oppression and constriction of the chest, with superficial hyperæmia and tenderness in the seat of tumor, local abstraction of blood by means of a few leeches, followed by a warm poultice, will give immediate relief. In cases of general or local venous congestion from pressure upon one of the principal veins, or of great oppression of breathing with hæmoptysis, bleeding from the arm, by reducing the volume of the blood and lowering vascular

\* *Dublin Medical Press*, April 19th, 1848.

† *Vide* case by Dr. J. S. Hughes, in *The Medical Press and Circular*, Oct. 15th, 1873.

‡ I have latterly used the compound solution of sulphate of morphia and atropia recommended by Dr. James Little (*Dublin Journal of Medical Science*, vol. liv.) m v of this solution contains  $\frac{1}{6}$  of a grain of morphia, and  $\frac{1}{120}$  of a grain of atropia.



The bleeding should be repeated from time to time, and the quantity should be regulated according to the degree of congestion and the urgency of dyspnoea. I think there are few cases in which, even in the advanced stages of aneurism, the patient will not, in such circumstances, derive benefit from the loss of six to eight ounces of blood. In these cases, likewise, and with the same object in view, hydragogue aperients should be occasionally administered, whilst the allowance of liquids to be drunk should be restricted as far as practicable. For the purpose of insuring sleep, opium, chloral hydrate, or bromide of potassium, should be given in adequate doses, if necessary. I have found a combination of the two latter, in doses of xv grs. to xx grs. of each, more efficacious than either given alone.

The curative treatment of aneurism should be directed to the twofold object of effecting closure of the sac by deposition of fibrin, and arresting further arterial change by promoting general nutrition. In the system of Valsalva and Albertini the latter object was lost sight of. Hence, irrespectively of its irksomeness, and the consequent difficulty of pursuing it for a sufficient length of time, this system of treatment, which consisted in copious and repeated bleeding from the arm, active purgation, and a starvation diet, with absolute rest in the recumbent posture, was defective even in its conception.\* M. Bertin was the first writer who took exception to this system, and ventured to question the success claimed for it in the treatment of aneurism of the aorta. He adopted the modified plan of leeching, enjoining absolute repose of body and mind, and restricting within moderate limits the diet of the patient.†

Doctor Proudfoot, in the same year (1824), published two cases, in the treatment of which leeching and purgation were the only portions of Valsalva's system adopted.‡

Doctors Graves and Stokes, whilst recommending this system of treatment in the early stages of thoracic aneurism, suggested, in all other cases, the opposite plan in regard to diet, which had been

\* Vide *Morgagni de Sedibus et Causis Morborum*, lib. ii., epist. xvii., § 30 et 31.

† *Traité des Maladies du Cœur*, p. 151.

‡ *Edinburgh Medical and Surgical Journal*, vol. xxii.



has more recently objected to a lowering plan of treatment on several grounds: The advanced age of most of the subjects of aneurism; and the feeble state of nutrition, the irritability of the heart, and the hypinotic state of the blood produced by it.

With the view of promoting a deposition of stratified fibrin within the sac, and its subsequent solidification, measures must be taken to ensure cardiac and vascular quiescence as the first condition of success. All other means are subordinate to these. Hence, "Tufnell's plan," under which the patient is strictly confined to the recumbent posture for two or three months, supplies the most essential condition of a rational system of treatment.

According to Bellingham, to whom the merit of conceiving this plan is due, and Tufnell, who has developed and applied it with considerable success, the patient's diet and drink must be likewise restricted within prescribed limits.

Bellingham would limit the patient to three meals a-day: the morning and evening meal to consist of two ounces of liquid and four of solid nutriment; the mid-day meal, of four ounces of liquid, with from four to six ounces of solid. The liquid may consist of milk or tea, the solid of bread, and the mid-day meal of bread and meat in equal quantity.† This dietary must be strictly persevered in for a month or six weeks, whilst repose of mind and body is, as far as possible, to be maintained. The bowels should be kept free, but systematic purgation is deprecated.

Mr. Tufnell has, with considerable success, pursued this plan of treatment in a still more rigid form.‡ The patient is strictly confined to the horizontal posture for a period varying from eight to thirteen weeks, according to the effect upon the aneurism. During this period, the only change of posture permitted is turning slowly upon either side, or upon the face, whilst, by special arrangement of the bed, the bowels and bladder may be evacuated without disturbance of the body. The following scale of diet is, as far as practicable, to be carried out, viz.: For breakfast, two ounces of white bread and butter, with two ounces of cocoa or milk; for dinner, three ounces of meat, with three

\* *Dublin Hospital Reports*, vol. v., 1830.

† *A Treatise on the Diseases of the Heart*, 1857, p. 620.

‡ *The Successful Treatment of Internal Aneurism*, 1864.

charet, for supper, two ounces of bread and butter, and two ounces of milk or tea; the total amount of solid food allowed in the twenty-four hours being ten ounces, and of liquid food, eight ounces. After the contents of the sac have been in some degree solidified, or earlier if the patient prove restive under restriction, a more liberal scale of diet is allowed. As to medicine, sedatives, and especially opium, are given according to the urgency of pain, whilst moderate relaxation of the bowels is to be effected by means of ordinary aperients.

Mr. Tufnell has reported six examples of solidification of aortic aneurism under the foregoing plan of treatment, viz.: two of thoracic, and four of abdominal aneurism.

In a recent communication Mr. Tufnell has adduced, from his own practice, three additional examples of the successful treatment of aneurism by the same means, viz.: two of aneurism of the abdominal aorta, which were solidified in thirty-seven and twenty-one days, respectively; and one of popliteal aneurism cured in twelve days.\*

The importance of the principle involved in this mode of treating internal aneurism, and the encouraging results obtained from its adoption, must be my apology for the foregoing lengthened notice.

As an element in the curative treatment of aneurism, general blood-letting is likewise occasionally demanded. Vascular plethora and extreme tension of the sac may be most promptly corrected by bleeding to the amount of ten to twelve ounces, and should be practised where not absolutely forbidden by the debility of the patient. Dr. D. Gatzuc has shown experimentally that the withdrawal of blood from a vein or artery retards the current and reduces the blood-pressure, and in proportion to the quantity drawn.† Therefore, whether for the purpose of averting rupture of the sac, promoting deposition of fibrin within it, or of alleviating urgent symptoms arising from excentric pressure, an occasional bleeding by venesection may be resorted to with advantage in suitable cases.

Where this seems inadmissible, the object may be accom-

\* *Medico-Chirurgical Transactions*, vol. xxxix., second series, 1874.

† *British and Foreign Medico-Chirurgical Review*, April, 1872.

where the sac is nearest to the surface. But should the aneurism project externally, the leeches should be applied, not over the tumor, but immediately around it.

The medicinal agents which have been used with the object of effecting or promoting fibrination in the sac, may be now briefly noticed.

Acetate of lead has been strongly recommended by Hope. An example of success by means of this agent, given in doses of grs. iv to viii, thrice daily, has been reported by Hoegh.\* Some doubt is, however, expressed by the author as to the accuracy of his diagnosis. Mr. T. Holmes has judiciously remarked that painters are notoriously prone to aneurism. I have given the acetate of lead a fair trial, but without apparent benefit.

Iodide of potassium has been long used in the treatment of aneurism. According to Dr. Walshe, it was first recommended by Bouillaud in 1859, and next by Chuckrubby in 1862. In 1869, Dr. George W. Balfour recorded several cases of aneurism successfully treated with the iodide,† and still more recently two additional examples of aneurism, cured by means of large doses of this drug, were reported by the same physician.‡ The aneurism in one of these cases was thoracic, and was cured after a treatment of twenty months. In the second case, both an innominate and an abdominal aneurism existed. The doses of the iodide given, varied from grs. x to grs. xv, thrice daily. A case of thoracic aneurism, successfully treated with the same agent, has been published by Dr. Dyce Duckworth.§

My own experience of the iodide of potassium in the treatment of aneurism is rather favourable. I have tried it in several cases in the doses recommended by Dr. Balfour; but in no instance have I given it for more than one month. I have found it to relieve pain in some instances, an effect which I attribute to its property of lowering blood-pressure, and so lessening the tension of the sac.

\* *Medical Press and Circular*, June 23rd, 1869, translated by W. D. Moore from *The Norsk Magazin*, 1868.

† *Edinburgh Medical Journal*, 1869.

‡ *Ibid.*, December, 1871.

§ *British Medical Journal*, August 23rd, 1873.



a view of summarizing the contents of the sac.

I have tried phosphorus, with the object of arresting vascular degeneration, but only in one case (No. 142), and in that instance the disease was too advanced to admit a hope of much benefit from medicine of any kind. At an earlier period of the disease it may, perhaps, be found useful.

Galvano-puncture has been used by several physicians: Ciniselli,† McCaul Anderson,‡ and Charlton Bastian.§ Complete success has attended this plan of treatment in the hands of Ciniselli and Dr. Anderson. Out of 12 cases in the practice of Ciniselli, 10 were reported as well within periods varying from twelve to twenty-one months after operation; relapse occurred in 2 of the cases, in one of these a second and successful operation was performed, the other died within eighteen months; death resulted directly from the operation in 4 cases. Another successful case by Ciniselli has been reported by Dr. Althaus.|| In Dr. Anderson's case, four operations were performed. The patient, though not perfectly cured, was reported as well seven months afterwards..

Mr. C. H. Moore and Dr. Murchison made the bold attempt to bring about coagulation by introducing several yards of iron wire into the sac. Death occurred on the fifth day after operation.¶

Where the tumor points externally and the sac is making progress towards the surface, a well-adjusted pad, strapped down over it by means of adhesive plaster, will not only relieve the pain arising from the tension of the skin, but will likewise retard the advance of the aneurism.

More than a reference to the surgical treatment of aneurism of the aorta by distal pressure or ligature, would manifestly be out of place in this work. For a full discussion of this subject, the reader is referred to the admirable and exhaustive lectures of Mr. Timothy Holmes.\*\*

\* *Berlin. Klin. Woch.*, No. 12, 1869; Abstract in *Dublin Journal*, May, 1870.

† Quoted by Mr. Holmes, *Lancet*, November 9th, 1872.

‡ *Lancet*, vol. i., 1873.

§ *British Medical Journal*, November 22nd, 1873.

|| *Treatise on Medical Electricity*.

¶ *Medico-Chirurgical Transactions*, vol. xlvii., 1864.

\*\* *On the Surgical Treatment of Aneurism*, *Lancet*, July 13th and 27th, 1872.



been reported by Mr. C. Heath. The case was one of aneurism involving the ascending and transverse portions of the arch of the aorta, and pressing on the left subclavian artery. A ligature was applied to the left carotid above the omo-hyoid muscle. The tumor receded, and all the previous signs disappeared. A year after the operation, the patient, a man aged forty-eight, was reported as having been, in the interim, actively employed as a farm-labourer. The prominence of the chest in the seat of tumor had then disappeared; dulness was gradually clearing up, and the man was virtually in perfect health.

Another successful case has been reported from the practice of the same surgeon.† In this case, which was one of aneurism of the arch of the aorta involving the origin of the arteria innominate, both the carotid and the subclavian artery of the right side were ligatured. The patient, a woman, left hospital convalescent three months afterwards; but, owing to the administration of an emetic, the aneurism again became active, and soon proved fatal.

A case in which the distal ligature was used for the cure of an innominate aneurism has been recorded by the late Mr. Hutton. Secondary hæmorrhage occurred, and the patient died by syncope on the seventy-sixth day after the operation.‡

As to the use of distal compression for the cure of an innominate aneurism, under the prolonged administration of chloroform, Mahomed declares that this procedure is not entirely free from danger. Amongst the consequences to be apprehended are apoplexy, of which he gives an example from compression of the right carotid for the cure of an aorto-innominate aneurism; retroversion of a diseased aortic valve; dilatation of the left ventricle, with aggravation of pre-existing mitral reflux; and rupture of the sac. Where left ventricular hypertrophy and general atheroma exist, compression, if attempted, should be made without chloroform, or a ligature should be applied.§

The following Table (XVI.) has been constructed from the records of the Pathological Society of Dublin.

\* *Clinical Society's Transactions*, vols. v. and vi.

† *British Medical Journal*, May 21st, 1870.

‡ *Proceedings of the Pathological Society of Dublin*, December 10th, 1842.

§ *Medical Times and Gazette*, August 9th, 1873.

1	Dr. Law	Male, 43	..	24 : Thoracic aorta	Good	Systolic	None	Systolic	Exhaust
2	Dr. Smith	Male, 47	..	24 : Transv. portion of arch	..	Systolic	None	None	Coma
3	Dr. Law	Male, 42	..	4 : Ascend. portion of arch	..	..	..	..	Coma
4	Dr. Lees	Female, 60	..	Ascend. portion of arch	..	..	..	..	Rupture to perica
5	Dr. O'Ferrall	Male, 45	..	Middle of transv. portion of arch	..	None	None	None	Asphyxi
6	Dr. J. C. Ferguson	Male	..	Ascend. portion of arch	..	..	..	..	Rupture to perica
7	Dr. Bigger	Soldier	..	Ascend. and part of transv. portion of arch	..	None	None	None	Exhaust
8	Dr. Greene	Male, 39	..	Ascend. portion of arch and abdominal aorta	Emaciated	Systol. in abdomen	None	Systolic	Exhaust
9	Dr. Lees	Female, 62	..	Transv. portion of arch	..	..	..	..	Rupt. in left lu
10	Dr. Stokes	Male, 39	Temperate	24 : Upper part of descend. aorta	..	Systolic	None	Systolic	Intercu: bronch
11	Sir P. Cramp-ton	Male	..	Junction of ascend. and transv. portion of arch	..	None	Systolic	Systolic	Syncope
12	Dr. Law	Slater, 92	Intemperate, fall from scaffolding	Left extremity of transverse portion	..	None	None	Systolic	Rupt. in left l & ple
13	Dr. McDowel	Male, 40	..	Transv. portion of arch	..	None	Systol. and diastol.	Systolic	Asphyxi
14	Sir D. Corrigan	Male, 32	..	2 : Upper part of ascend. and whole of transv. portion of arch	..	None	None	None	Rupt. in trach
15	Dr. O'Ferrall	Soldier, 36	Intemperate	Transv. portion of arch	Emaciated	None	None	None	Rupt. left b chus
16	Dr. McDowel	Porter, 35	..	10 days : Transv. portion of arch, engaging origin of arteria innominate	..	None	None	None	Asphyxi
17	Dr. J. F. Duncan	Bricklayer 40	Intemperate	4 : Ascend. portion of arch, involving arteria innominate	..	Systolic	None	Systolic	Coma
18	Dr. Stokes	Porter, 50	..	12 : Left extremity of transv. portion	Good	None	None	None	Rupt. tract
19	Dr. Mayne	Horse-dealer	Intemperate; kick from a horse on chest	9 : Com. of descend. portion of arch	..	None	None	None	Exhaust repeated morrb
20	Dr. Hudson	Male, 34	..	42 : Middle of thoracic aorta	..	None	None	None	Rupture to left and l
21	Dr. H. Kennedy	Male, 44	..	4 : Upper extremity of ascending portion of arch	..	None	None	Systolic	Exhaust
22	Mr. Symes	Police Constable	..	Left extrem. of transverse portion of arch	..	..	..	..	Rupture to left and into pleu Asphy
23	Dr. Mayne	Compositor, 42	Intemperate	24 : Inferior portion of thoracic aorta	..	Systolic	None	Systolic	
24	Dr. Gordon	Porter, 34	..	4 : Anterior sinus of Valsalva	..	Systolic	None	None	Asphy

Back. & laterally	None	..	Atheromat. in seat of aneurism	Six aneurisms.
Backward	None	..	..	..
Back. and to right	..	..	..	Aneurism occluded superior vena cava.
Up. & down. in course of aorta	Aortic and mitral	R. Normal L. Hypert. and dilated	Atheromatous	Hypertrophy and dilatation; dissecting aneurism; no previous history.
Backward	..	..	..	No dulness, impulse, murmur, or interruption of circulation; paroxysmal dyspnoea, bordering on asphyxia.
Backward and downward	..	..	..	Pressure on superior cava, and lividity of face and neck.
General expansion	Aortic	R. Dilated L. Dilated and hypertrophied	Atheromatous	Enlarged.
Forward in both	Aortic	..	Atheromatous	Double aneurism.
Forward, and to left	..	..	Atheromatous	No suspicion of aneurism; rupture into substance of left lung at apex, which was attached to sac.
Forward, and to right	..	..	..	Symptoms of aneurism well pronounced; also physical signs in upper left front. Both disappeared—former returning after six months; latter not returning.
Forward	None	Normal	Healthy except at seat of aneurism	Not hypertrophied. Effusion into pericardium.
Back. and to left	None	..	Atheromat. in seat of aneurism	Rupture into apex of left lung, followed by fatal hæmoptysis.
Back. and to right	None	Normal	..	Heart fatty; no stridor; asthenia.
Back. and to right	None	Normal	Atheromat. in seat of aneurism	No hypertrophy.
Back. and to left	..	..	..	No physical sign of aneurism, except loud ringing cough.
Down. back. & to left	None	Normal	Atheromatous	Not hypertrophied; fatty; no <i>bruit</i> , impulse, or laryngeal signs, but intense paroxysmal dyspnoea.
Forward and to right	Aortic	..	Atheromatous	Pressed on superior cava, causing cyanosis and cedema of upper half of body, and contraction of right pupil.
Back. and to right	None	Normal	Atheromatous	No hypertrophy; stridor, dyspnoea, or physical signs.
Forward and to left	..	..	..	No symptoms or signs. Aneurism was imbedded in apex of left lung, into which it had leaked.
Forward and to left	None	..	..	No special symptoms or signs. Fixed pain in back, and shooting pains through chest.
Forward and upward	None	Normal	Atheromat. in seat of aneurism	No hypertrophy. Symptoms were dysphagia, cough, a pulsating tumor in root of neck, and turgescence of superficial veins of chest.
Forward and to left	..	..	Atheromatous	Tumor involved in left lung. No suspicion of aneurism.
Forward and to left	Slight thick. of aortic valves, which were competent	..	Atheromat. in seat of aneurism	Not hypertrophied. Aneurism involved in left lung, into which it had leaked.
Downward and to left	Aortic ruptured	R. Dilated L. Dil. and hypertrophied	Atheromatous	Hypertrophy. Aneurism projecting into right ventricle, and opening into left ruptured ventricle through aortic valve.

25	Dr. Gordon	Male	..	48 : Two, viz. ; in com. of ascending portion of arch & of thoracic aorta	..	Systolic	None	Systolic	Exhausti
26	Dr. Banks	White-smith, 50	Temperate ; severe anvil work	90 : Upper portion of ascend. aorta	..	None	Two	Systolic and diastolic	Exhausti
27	Mr. Symes	Male, 50	..	Entire extent of aorta	..	..	..	..	..
28	Dr. Banks	Male, 46	..	36 : Left extremity of transverse portion of arch	..	None	Two	Systolic and diastolic	Syncope
29	Dr. Minchin	Female, 44	..	11 : Middle of transv. portion of arch	..	Systolic	None	Systolic	Rupture
30	Dr. E. Hamilton	Police Constable	Severe duty	Entire of transv. portion of arch	Emaciated	None	None	Systolic	ternal Rupture to trachea Exhausti
31	Dr. Jennings	Dischg'd. Soldier, 36	..	36 : One of the sinuses of Valsalva	..	None	None	None	Exhausti
32	Dr. Banks	Soldier, 28	..	Com. of transverse portion of arch	Good	None	None	None	Rupture left ple
33	Mr. Porter	Male, 68	..	24 : Descending portion of arch	..	None	None	None	Asthenia
34	Dr. E. Bennett	Coal-porter, 40	Intemperate	One of the sinuses of Valsalva	..	Systolic	None	Systolic	Bronchi
35	Dr. Banks	Shoe-maker, 46	Temperate	6 : Whole of transv. portion of arch	Worn	None	Two	Systolic	Syncope
36	Dr. Finny	Soldier	Temperate	One of the sinuses of Valsalva	Good	Systolic	None	Systolic	Rupture to peric Exhausti
37	Dr. Jennings	Labourer, 44	Strain	120 : Ascending portion of arch	..	None	None	Systolic and diastolic	Exhausti
38	Dr. Richardson	Female, 40	..	6 : Termination of ascend. and whole of transv. portion, including arteria innominata	..	None	None	Systolic	Pleuro-pneum
39	Mr. Porter	Shoe-maker, 37	Intemperate ; great physical exertion	Ascending portion of arch.	Good	None	None	None	Rupture to peric
40	Dr. H. Kennedy	Female, 60	..	24 : Junction of ascend. and transv. portion of arch	..	None	None	Systolic	Asthenia
41	Dr. Law	Butler, 60	Intemperate	Entire of ascending portion of arch	Good	None	None	None	Syncopa
42	Dr. Stokes	Gentleman 54	Temperate ; physical effort	24 hours ; ascending portion of arch	Good	None	None	None	Rupture to peric
43	Dr. Quinlan	Male, 40	..	Junction of transv. and ascend portion of arch	..	None	None	None	Syncopa
44	Dr. Lyons.	Male, 45	Intemperate ; blow upon chest	36 : Com. of ascend. portion of arch	..	None	None	None	Rupture to peric
45	Dr. Lyons	Sailor, 36	..	8 : Ascend. portion of arch	..	Diastolic	Systolic	Systolic and diastolic	Rupture to circumscribe cavity i pleura Rupture to peric
46	Dr. Lyons	Labourer	..	8 days : two aneurisms, one in up. part of ascending port. of arch, and the other at root of vessel	..	Systolic	None	None	Rupture to peric
47	Dr. W. Moore	Cardriver	Intemperate	8 : Com. of thoracic aorta	Emaciated	None	None	None	Rupt. left bronch
48	Dr. MacSwiney	Male	..	Some months ; thoracic aorta	..	None	None	None	Rupt. right p



The first for. & down., & the second for. & to left	Aortic	..	Atheromatous	Two aneurisms.
Forward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied. Constant pain in chest, neck, and arm, with severe paroxysms.
Course of vessel	Aortic and mitral	R. Not stated L. Dilated and hypertrophied	Atheromatous	Hypertrophy. Subject sent for dissection. No history; dissecting aneurism from root of aorta to left extremity of iliac artery, into which stream re-entered.
Forward and backward	None	Normal	Atheromat. in seat of aneurism	Not hypertro.; contraction of left pupil; great variation in direction of growth.
Forward and upward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied; external hæmorrhage by dislodgment of fibrinous plug.
Backward	None	Normal	Atheromatous	Not hypertrophied. Occlusion of right subclavian artery.
Upward and to right	Aortic and mitral	Dilated and hypertrophied	Atheromatous	Hypertrophied. Advanced valvular disease.
Forward and to left	None	Normal	Atheromatous	Not hypertrophied. No suspicion of aneurism, and no symptoms except very slight cough.
..	..	..	Atheromatous	Had had popliteal and axillary aneurism.
Downward into R. ventricle	None	R. Normal L. Hypertro.	Atheromat. at seat of aneurism	Hypertrophy. Aneurism projected into right ventricle.
Forward	Aortic & mitral	Dilated	Atheromatous	Dilated and fatty.
Upward	None	R. Normal L. Hypertro.	Healthy	Hypertrophy; aneurism, size of walnut
Forward	None	Dilated	Atheromatous	Heart dilated and fatty. Atrophy of brain and recurrent paralysis.
General expansion	None	Normal	Atheromatous	No hypertrophy; heart fatty.
Forward	..	..	Atheromatous in seat of aneurism	Two aneurisms. Man had run a race a few days before death. No suspicion of aneurism.
Forward and to right	None	Hypertrophied	Atheromatous	General hypertrophy.
Forward and to right	None	Normal	Atheromatous	Not hypertrophied, soft and fatty.
Course of vessel	None	Normal	Atheromatous	Not hypertro. Aortic and pulmonic valves atrophied and cribriform; heart fatty; "dissecting" aneurism bursting into pericardium at root of aorta. The patient (Dr. Ball) survived the rupture of internal and middle coats 108 hours; rent in vessel, two-thirds round.
Forward and to left	None	..	Atheromat. in seat of aneurism	Hypertrophied.
Course of vessel	None	..	Atheromatous	"Dissecting" aneurism. Rent in coats of vessel two-thirds round, probably occurred three years before.
Forward	None	..	Atheromat. in seat of aneurism	Double murmur in aorta from inadequacy, the result of dilatation of the vessel.
Upper forward, and lower in course of vessel	None	..	Atheromatous	The lower was a "dissecting" aneurism.
Forward	None	..	Atheromatous	Aneurism not suspected.
Forward and to right	..	..	Atheromatous	Heart fatty. No history, as man was moribund when admitted.

49	Dr. Eames	Male	Temperate	4: Three aneurisms; one from right anterior sinus of Valsalva, and two in ascend. port. of arch above former	..	Systolic	None	None	Rupture second aneurism to trachea
50	Dr. Law	Labourer, 40	Temperate	4: Posterior sinus of Valsalva	..	Systolic and diastolic	None	None	Coma
51	Mr. J. Hamilton	Pensioner, 39	Strain in lifting weight	8: Transv. port. of arch at middle	..	None	Two	Systolic	Asphyxia
52	Surgeon J. S. Chartres	Soldier, 36	Intemperate; crush by a horse falling on him	24: Com. of thoracic aorta	Emaciated	None	None	None	Exhaust
53	Dr. McDowel	Poor woman, 65	..	Commencement of ascending portion of arch	Good	None	None	None	Rupture to pericardium
54	Dr. MacSwiney	Poor woman, 40	..	24: Entire arch	..	None	Two	None	Bronchitis
55	Dr. A. W. Foot	Shoemaker, 40	..	3: Com. of ascend. and mid. of transv. portion of arch	..	Systolic in lower aneur.	None	None	Bronchitis
56	Dr. T. E. Little	Poor woman, 38	Intemperate	12: Ascending portion of arch	Good	None	None	Systolic	..
57	Dr. Lyons	Groom, 42	Kick from a horse on the chest	18: Ascend. portion of arch, and lower portion of thoracic aorta	Good	None	Two	Systolic and diastolic	Exhaust
58	Dr. Finny	Dairyman, 33	Intemperate	12: Ascend. portion of arch	Emaciated	Systolic	None	None	Exhaust
59	Dr. G. F. Duffey	Soldier, 30	Temperate	30: Ascend. portion of arch above left valve	Good	..	..	..	Rupture to pericardium
60	Dr. Hayden	Cabdriver, 33	Intemperate; severe blow on back	4: Ascend. portion of arch	Good	None	Two	Systolic and diastolic	Rupture to pleura
61	Dr. Hayden	Hatter, 34	Intemperate	9: Superior thoracic	Fair	None	None	None	Rupture left ventricle to pericardium
62	Dr. Hayden	Cabdriver, 28	Intemperate; strain in lifting a weight	39: Inferior thoracic	Fair	Systolic	None	Single and systolic	Asthma
63	Dr. Hayden, per Dr. Stokes	Player upon a wind instrument, 46	..	7 weeks: Ascending portion of arch	Good	Systolic and diastolic	None	None	Slow pharynx
64	Dr. Hayden	Egg-packer, 33	Intemperate; carrying heavy load	6: Left extremity of arch	Good	None	None	None	Asthma
65	Dr. Hayden	Architect, 56	Not intemperate	12: Ascend. & transv. portion of arch	Good	None	None	None	Rupture pericardium
66	Dr. Hayden	Housekeeper, 31	Temperate	1: Transverse	Good	None	None	None	Asphyxia
67	Dr. Hayden	Mason, 33	Intemperate; strain in lifting a weight	5 weeks: Com. of ascending portion of arch	Good	Systolic	None	Systolic	Asthma
68	Dr. Hayden	Labourer, 54	Intemperate	5 weeks: Ascending portion of arch	Good	None	Two	Double	Slow pharynx

Forward and downward	None	Normal	Atheromatous	Not hypertrophied. Liver depressed by lower aneurism.
Downward, forward, and to right	None	R. Dilated L. Dilated and hypertrophied	Atheromatous	Hypertro. and dilat. Depression of liver and jaundice. Aneurism projected into right auricle, and, by blocking venæ cavæ, caused general congestion and dropsy.
Forward, upward, and to right	None	..	..	Not hypertro. Aneurism had ascended on right side of neck, in course of carotid artery.
Forward, and to left	None	Normal	Atheromatous	Not hypertrophied.
Course of vessel	..	..	Atheromat. in seat of aneurism	Dissecting aneurism; commenced by rent of internal coat one inch above valves, extended some distance upwards and opened below into pericard. No history, and no opportunity for physical examination.
Backward and to right	None	Reduced in capacity	Atheromatous	Atrophied. Bilobed aneurism.
General dilat. of inferior, and back-growth of superior aneurism	None	R. Dilated L. Dilated and hypertrophied	Atheromatous	Hypertrophied. Two aneurisms.
Forward and to left	None	..	Atheromat. in seat of aneurism	Aneurism extending forward and to left side, pressing on root of left lung, and causing absorption of ribs beneath left clavicle.
Superior forw. and inferior backward	Aortic slightly	..	Atheromatous	Two aneurisms.
Upward and to both sides	None	R. Dilated and hypertrophied L. Atrophied	Atheromatous	Left ventricle atrophied; the patient was in an advanced stage of phthisis.
General expansion	None	None	Highly atheromatous	No symptoms, and therefore no suspicion of aneurism; the man dropt down in the act of pumping water for the regiment.
Forward and to right	Slight thickening of aortic	R. Normal L. Concent. hypertrophy	Atheromatous	Not enlarged. Fatty accumulation. Severe and paroxysmal pain in right side of chest and arm, relieved by leeching.
Forward	None	Normal	Slightly atheromatous	Less than normal in size. Fatty accumulation. Aneurism, latent as to physical signs, but diagnosed from negative and rational evidence. Pneumonia, without physical signs.
Forward chiefly	None	Normal	Healthy, except at seat of aneurism	Atrophy. Severe paroxysmal pain in back and abdomen; large false aneurism
Downward and to right	None	Normal	Highly atheromatous	Not hypertrophied. Aneurism of ascending aorta opening into right ventricle. (See Case 69, p. 759.)
Backward, and to right	None	Normal	Atheromat. in seat of aneurism	Slightly enlarged (11 ozs.)
Upward, downward, and laterally	None	R. Slight hypertrophy L. Slight hypertrophy and dilatation	Highly atheromatous	Slightly enlarged, and fatty accumulation. Large aneurism engaging entire arch, but latent.
Upward & backward	None	Normal	..	No hypertrophy. Fatty accumulation.
Upward & backward	None	Normal	Atheromat. in seat of aneurism	Not hypertrophied.
Forward, up. and backward	None	R. Dilated L. Hypertro.	Highly atheromatous	Hypertrophy of left ventricle, and fatty accumulation.



to submit the following cases.

CASE CXXXV.—*Blow on Back ; Eighteen Months later, Pain in the Right Side of the Chest, followed by Pulsating Tumor beneath the Right Clavicle ; Double Impulse and Sound in Tumor ; Respiration Normal ; Murmur at Left Base ; Severe Pain in Scapular Region relieved by Leeching and Morphia ; Œdema of Right Side, and Effusion into Right Pleura ; Sudden Death by Rupture of Sac and Extravasation into Right Pleural Cavity. Aneurism connected with Ascending Portion of the Arch of the Aorta, and containing Loose Clot ; Inferior Lobe of Right Lung Condensed by Effusion ; Slight Effusion into Pericardium ; Heart Fatty on Surface, but structurally Sound ; Aortic Valves slightly Thickened ; Left Ventricle in a state of Concentric Hypertrophy ; Aorta Atheromatous.*

William R., aged thirty-three years, of very intemperate habits, was received into hospital on the 12th day of February, 1866. Two years previously he had been knocked down by a cab whilst intoxicated. He received the blow upon the back, and was stunned by it, but he quickly recovered, and felt no further inconvenience from the accident till six weeks before admittance, when he was seized with pain in the upper part of the right side of chest ; three weeks later he observed a pulsating swelling in the same situation. When he came under my notice, he was pale, but well nourished, and breathing freely and without difficulty in the recumbent posture. Pulse 84, regular, moderately full, and equal. Pupils normal and equal ; no dysphagia. His only complaint was, of pain in the chest extending to the right shoulder. A prominence existed on the superior and anterior portion of the right side of the chest ; it was semi-globular, closely resembling the female breast in figure, and about three inches in diameter at the base ; it extended from a line half an inch beneath the clavicle, nearly to the level of the nipple, and from the right margin of the sternum to the middle of the clavicle. This swelling projected about an inch and a-half, was dull on percussion, and yielded a double shock ; the first,



diastolic in rhythm, and presented the character of a back-stroke during recoil of the tumor. The primary shock was stronger on the outer than on the inner side of the swelling, and was here distinctly excentric. No respiratory sound was audible over the projecting portion of the chest; but two sounds, remarkably clear, free from murmur, and synchronous with those of the heart, were heard. Over the remainder of the right side, and over the entire of the left side of the chest, percussion and respiratory sounds were normal.

The apex of the heart pulsated in the sixth intercostal space half an inch outside the nipple-line; precordial dulness was not extended. The first sound of the heart, as heard at the apex, was soft and prolonged; at the left base it was accompanied by a sharp "whiffing" murmur. This murmur was loudest in the area of the pulmonic orifice, but it was diffused universally over the left front of the chest; it could not be heard to the right of the mesial line, nor was it audible at the acme of inspiration or in the early period of expiration. Posteriorly, respiration was somewhat louder on the left than on the right side, and over both sides a double sound, synchronous with those of the heart and tumor, was heard. Neither impulse nor murmur could be detected posteriorly, nor was there anywhere along the spine a point of tenderness to pressure.

The diagnosis was not difficult. The sac of the aneurism was partially filled with fluid blood, for it was possible, by pressure, to reduce its volume considerably.

Whilst under treatment, the man complained occasionally of severe pain in the situation of the tumor, and extending to the right scapula. This pain, which was of a darting and intermittent character, deprived him for the time, of sleep; it was, however, always promptly and effectually controlled by the application of one or two leeches over the seat of its greatest intensity, followed by a draught containing ℥xl of the pharmacopœial solution of hydrochlorate of morphia. The pulse varied from 78 to 96, and was regular. Decubitus was dexter, and hence, there was slight œdema of the right shoulder and axilla. The carotid arteries pulsated visibly, and yielded a double sound but no murmur.

mur on gentle pressure.

The man's condition underwent very little change between the 12th of February and the 14th of March, during which period he had been taking iron. On the last mentioned day, I remarked that the aneurism had advanced considerably in the direction forwards and outwards; it had become more solid, and its pulsation was stronger. The cedema of the right side now extended to the forearm, where the patient complained of sharp pain. Pulse 96. The iron was now stopped, digitalis was given, and two leeches were applied in the right axilla.

On the 19th, the pain was so severe as to induce copious perspiration; breathing was embarrassed even in the sitting posture, and the tumor had become more prominent.

On the 23rd the lower portion of the right side was dull, and respiration here was feeble and slightly bronchial.

At half-past six o'clock on the morning of the 25th, he called for his medicine, declaring he had had an unusually good night and felt much better; in the act of sitting up in bed he suddenly complained of agonizing pain in the abdomen, cried out that he was dying, was agitated, and became deadly pale; the tumor immediately ceased to pulsate, and the man died of syncope within thirty minutes.

The body was examined twenty-eight hours after death. The right side, measured over the prominence, exceeded the left in girth by three inches and a-half. The right pleural cavity was full of coagulated blood and serum; the clots alone weighed three pounds. On removing the blood, an enormous aneurism, as large as the head of a new-born infant, was seen to occupy the upper portion of the pleural cavity. It sprang from the upper part of the middle mediastinum, projected forwards and outwards, contracting adhesions to, and thrusting forward, the anterior wall of the chest, through which it had ultimately protruded by erosion of the second, third, and fourth ribs.

The aneurism was invested by the pleura in the greater portion of its circumference, and was unsupported, except at the root and inferiorly, where the middle lobe of the lung was attached to it. Below and behind the point of attachment just

through which two fingers might be passed; from this opening projected a large and jagged mass of partially decolorized clot. The superior and middle lobes of the lung were firmly attached to, and expanded upon the tumor, but they contained some air; the inferior lobe was entirely devoid of air, of a dark slate-colour, and pressed into the inferior and posterior portion of the pleural cavity.

The left lung was vascular on the surface, and partially emphysematous on the anterior margin, but otherwise healthy.

The pericardium contained about two ounces of serum; the heart presented a good deal of superficial fat at the base, and a thick layer in front of the ascending portion of the arch of the aorta.

On the right auricular appendix there was a large "milk-spot," and another existed in the usual situation on the right ventricle. A nodule of lymph, as large as a pea, was found attached to the left ventricle near the apex. The right chambers were normal; the left ventricle was much thickened, but diminished in capacity. The valves were all healthy, except those of the aorta, which were thickened and slightly incompetent.

The lining membrane of the aorta was rugous, and of a dirty yellow colour; a circular opening, which admitted the points of three fingers, existed in the right wall of the ascending portion of the vessel, one inch below the origin of the arteria innominata, and led into the large aneurism previously described. The sac contained a good deal of coagulum, which was attached to the anterior wall, and projected through the rent on its inferior surface. This rent was distant about two inches from the orifice of communication with the aorta.

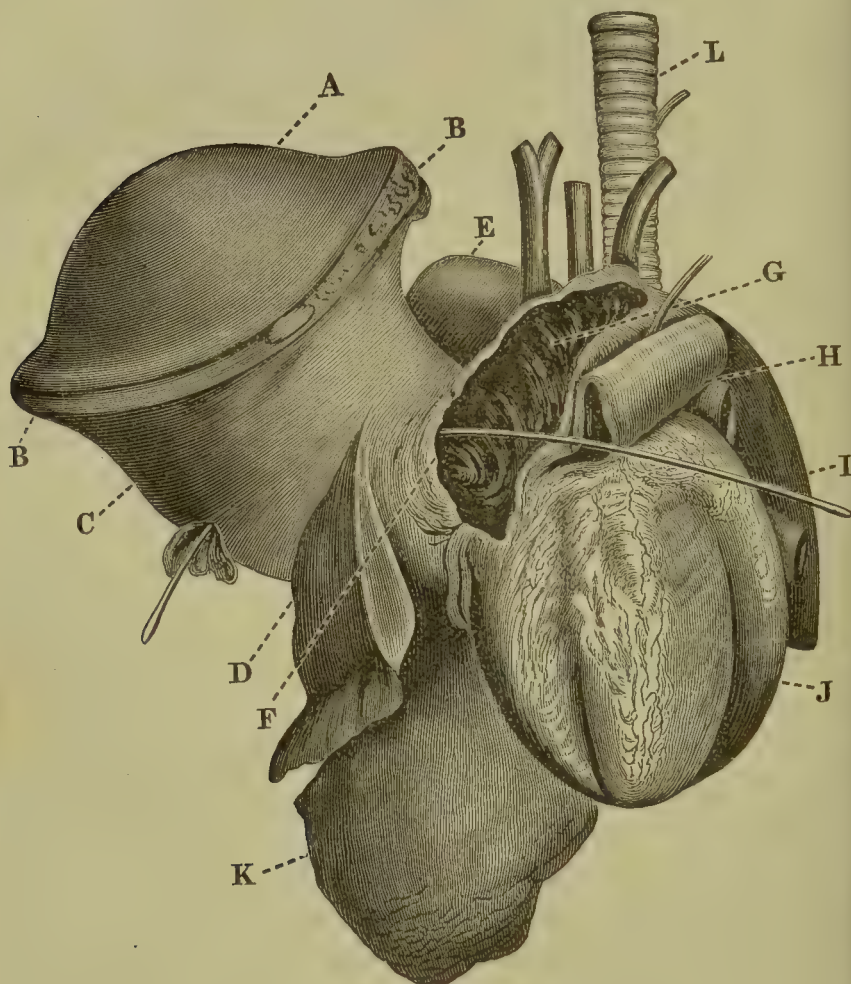
The pulmonary artery and its valves were healthy, as were likewise the primary branches of the aorta. The œsophagus, the descending cava and its tributaries, and the right bronchus, had escaped pressure; the pneumogastric nerves seemed much thickened.

The result of physical examination on the 23rd proved the existence, at that date, of serous effusion into the right pleura.



found in the pleura, by extravasation from the ruptured sac of the aneurism.\* The subjoined woodcut (Fig. 69) from a coloured drawing by Mr. W. Laffan, a student of medicine, exhibits the aneurism and the adjacent parts in their relative position.

FIG. LXIX.



Aneurism of ascending portion of arch of aorta.

William R.

\* Vide *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., p. 32, new series. I no longer admit, as I then stated, the existence of concentric hypertrophy of the left ventricle as a pathological entity, nor hypertrophy of that chamber as a consequence of aneurism of the aorta.



muscles having been removed by dissection.

- BB Section of sternum and ribs encircling the projecting aneurism.
- C Intra-thoracic portion of aneurism, exhibiting a rent in its posterior inferior wall, by which the fatal hæmorrhage had occurred, and through which a jagged clot and the end of a bent probe are seen to project.
- D Middle lobe of right lung adherent to aneurism.
- E Apex of right lung projecting above and behind aneurism.
- F Aperture in aorta leading into aneurism. A bent probe is represented as passed through the openings of entrance and exit of the aneurism.
- G Aorta laid open by removal of its anterior wall, exhibiting the atheromatous condition of its coats, and the great vessels arising from it.
- H Left pulmonary artery and bronchus; the pneumogastric nerve is seen coursing down behind them.
- I Descending portion of arch, and thoracic aorta, crossed by one of the left pulmonary veins.
- J The heart, exhibiting much fat on its anterior surface.
- K Inferior lobe of right lung compressed by the blood effused in the fatal hæmorrhage.
- L The trachea and left recurrent nerve.

The following measurements of the left ventricle were obtained ninety-six hours after death :

*Thickness of Walls.*

Near apex	...	...	...	...	$\frac{3}{4}$ inch.
Middle portion	...	...	...	...	$\frac{13}{16}$ "
Near base	...	...	...	...	$\frac{3}{4}$ "

*Dimensions of Cavity.*

Length from root of mitral valve to apex	...	...	2 inches.
Width at central portion, measured from septum	...	$\frac{1}{2}$	"
Width at base	do.	do.	$\frac{3}{4}$ "

It thus appears that the left ventricle was in a state of concentric hypertrophy. The increased growth of the walls was due to functional compensation, rendered necessary by the atheromatous change of the aorta which had preceded the development of aneurism; whilst the diminution of capacity depended upon spasm of the left ventricle, excited by the nervous shock arising from rupture of the aneurism, and favoured by the sudden withdrawal of vascular resistance in front.

*Left Side; Defective Motion and Absence of Respiration on Left Side, and subsequently Pneumonic Solidification of the Left Lung without the usual Physical Signs; Slight and Temporary Dysphagia; Cough not Laryngeal; No Stridor; Temporary Improvement; Sudden Death by Rupture of Sac simultaneously into Left Bronchus and Œsophagus. Aneurism connected with Upper Portion of Descending Thoracic Aorta, and pressing upon Left Bronchus; Aorta Atheromatous; Effusion into Pericardium; Heart Fatty on Surface but less than average in size, its Walls and Valves Healthy; Left Lung in Third Stage of Pneumonia.*

John S., a hatter, aged thirty-four years, temperate, was received into hospital for me on the 19th of September, 1868. He stated that, nine months previously, he had been attacked with pain shooting across the front of his chest, and extending downwards on both sides; four months later, this pain became fixed in the left side. He had continued at his business till the date of his admission. He lay habitually on the left side, and had never spat blood.

State on admittance: Patient pale; no cough. Pulse 96, regular, and equal in both arms; pupils normal and equal. He complained of a dull aching pain on the left side, but shifting occasionally to the epigastrium. The right temporal artery was full and tortuous, and pulsated strongly; whilst the left temporal artery pulsated feebly, and was not visible. A corresponding difference was observable in regard to the pulsation of the carotid arteries on the two sides. Respiration tranquil.

Both sides of the chest were perfectly and equally clear on percussion; but motion was defective on the left side, and respiration was nowhere audible on that side, except at the sternoclavicular articulation, and in the supra spinous fossa, in both of which situations an interrupted and harsh respiratory sound was heard. Respiration was puerile on the right side, and vocal vibration existed on both sides. No venous engorgement; no dysphonia, stridor, or dysphagia.

Treatment: Dry-cupping, and subsequently a few leeches at

tract of opium at night.

On the 22nd October the report was: Slept well and was free from pain last night; can now lie on either side; no change in percussion or respiratory phenomena. No impulse anywhere perceptible over the chest, except in the cardiac region, where it is normal. He was discharged on the 31st of October, nearly free from pain, and much improved in his general health.

I visited him at his lodgings on the 28th of November. During the preceding week he had been suffering from teasing cough attended with slight mucous expectoration, and had on several occasions vomited his food half an hour after taking it. He likewise felt obstruction in swallowing, above the pit of the stomach. The face was of a leaden tint. Right temporal artery full and tortuous; left not so. Pupils normal and equal. Pulse 108, weak but regular, and equal in the right and left arms. (See Figs. LXX. and LXXI.) The left infra-clavicular space,

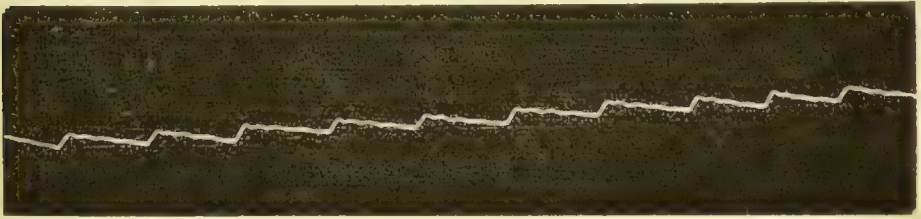


FIG. LXX.

Aneurism of descending aorta. Right radial pulse. John S.

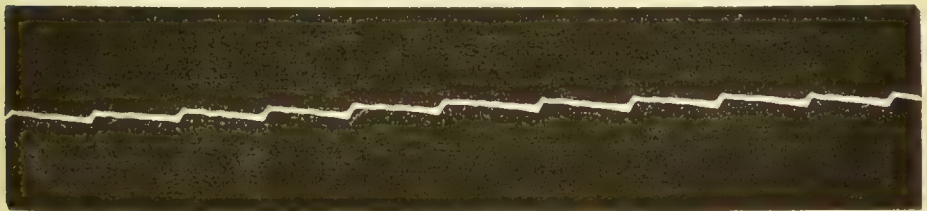


FIG. LXXI.

Aneurism of descending aorta. Left radial pulse. John S.

for a distance of two inches and a-half downwards, yielded the cracked-pot sound with muffled tympanitic resonance on percussion; below this level, as far as the nipple-line, there was comparative resonance, whilst over the remainder of the side



where audible on the left side, except in the interscapular space, where it was feeble and bronchial. In the first intercostal space on the left side, close to the sternum, a distinct impulse was felt during forced expiration, over a space of one inch in diameter. To have gr. iss of the aqueous extract of opium at night.

He was readmitted on the 3rd of December. The apex-pulsation of the heart, which was very feeble, was then felt in the fifth intercostal space, whilst in the third and fourth interspaces, an inch and a-half inside the nipple-line, a strong pulsation, synchronous with that of the apex, was felt; the sounds of the heart were normal, but in the last mentioned situation the second sound was double. Both sounds were very faint in the course of the aorta. There was complete and uniform dulness of the left side as far as the left sterno-clavicular line. To the same extent vocal vibration was absent; respiratory sound was likewise in abeyance except in the left interscapular space, where it was bronchial, and at the left sterno-clavicular joint, where transmitted tracheal breathing was heard. Motion of left side imperfect. No abnormal impulse was anywhere perceptible. There was teasing cough, which was slightly laryngeal.

On the 15th, his condition had undergone improvement. The iron mixture and an anodyne at night had been continued. He walked about the corridor in the early part of that day, dined upon a chop at 1 o'clock, and swallowed without difficulty; he complained of no pain. At 9.30 o'clock, whilst in bed, he uttered a loud scream, and immediately afterwards vomited a large quantity of blood, and whilst in the act of reaching for a basin to the adjacent stand, he fell upon the floor; here, a few seconds afterwards, he was found in a state of syncope, but still conscious, and surrounded by a pool of florid and partially coagulated blood. He died fifteen minutes subsequently.

On examination of the body, the left lung was found to be solid throughout, except to a slight extent along the anterior edge, which was emphysematous and slightly congested; elsewhere the lung was hepatized, and of a marble tint; on section it was found to be infiltrated with pus. The right lung was voluminous and partially emphysematous; there was no appear-



pericardium contained about six ounces of serum. The heart was below the average size of the organ in the adult male, and was covered by a thick layer of fat. The chambers were all empty, and the valves were normal. The aorta was slightly atheromatous throughout the arch, and the left carotid was compressed at its origin by a large and hard lymphatic gland. Several glands similarly altered were found at the roots of the lungs.

Two inches below the origin of the left subclavian artery there was a large rent in the aorta, extending over three-fourths of its circumference, and leading into an aneurism as large as an orange. The interior of the sac was lined by laminae of dense fibrin, half an inch thick, except anteriorly and to the right side where the mediastinal pleura alone formed its wall. Here a communication had been established between the aneurism and the left bronchus by an opening of the size of a pea; this opening presented jagged and sloughy edges, and was partially plugged by a shred of fibrin which was connected with the laminae within the sac. On the right wall of the sac, and half an inch distant from the former opening, another, which was as large as a goose-quill, and quite patulous, existed. This latter opening led into the œsophagus, the mucous membrane of which was eroded to some distance around it. The appearance presented on the œsophageal aspect was that of a funnel, of which the small end was formed by the aperture leading from the bottom of the ulcer into the sac.

The diagnosis of aneurism of the descending portion of the arch, or of the upper portion of the descending thoracic aorta, pressing upon the left bronchus, was made at an early period of the case.

The temporary pressure upon the œsophagus is worthy of notice. It is probable that, for a week preceding the 3rd of December, when the man complained of obstruction in swallowing, accompanied by pain "above the pit of the stomach," the mucous membrane of the œsophagus had been undergoing erosion over the prominence caused by the ingrowth of the aneurism.

From the absence of hæmoptysis and hæmatemesis preceding the fatal hæmorrhage, it must be presumed that no communica-

during this period; whence it would follow, that the opening into the left bronchus, and that into the œsophagus, had been suddenly and simultaneously established a quarter of an hour before death. The existence of an effective plug in the opening which led into the bronchus, renders it more than probable that the fatal hæmorrhage occurred through the œsophagus.\*

CASE CXXXVII.—*Patient Intemperate; Severe Strain of the Back followed by Dorsal Pain; Two Years later, Pain of a Paroxysmal Character in Epigastrium, Sides, and Back, and a Fixed Pain in Left Gluteal Region; Systolic Bellows-murmur at Epigastrium, and occasionally a Diastolic-murmur in the same situation, but no Impulse; Relief under Ordinary Treatment; Return of Severe Paroxysmal Pain in Left Side of Abdomen, and extending to the Left Hip and Knee; Tenderness and Murmur over Dorsal Spine; Excruciating Pain in Back which was relieved by hypodermic use of Morphia; Enlargement of Left Side; Absolute Dulness of its Lower Portion, with Hæmoptysis and Heaving Impulse; Upward Displacement of Heart, and Forward Displacement of Liver; Tympanitic Resonance of Apex of Left Lung; Partial Suppression of Urine; Paroxysmal Dyspnœa; Death by Exhaustion. Autopsy: Large False Aneurism arising from Inferior Portion of Thoracic Aorta, occupying nearly the entire Left Pleural Cavity, and extending Beneath the Diaphragm as far as the Left Kidney; Erosion of Three of the Dorsal Vertebrae.*

William F., aged twenty-eight years, a cabman of intemperate habits, first came under my notice about January, 1866, suffering from chronic laryngeal symptoms, manifestly the result of his irregular life. After a short course of treatment in hospital he was restored to his ordinary health, and discharged. Shortly afterwards he strained his back in lifting his cab, and then, for the first time, complained of pain in the lower dorsal spine. He

\* Vide *Proceedings of the Pathological Society of Dublin*, new series, vol. iv., part i., p. 3.

the 22nd of June, 1868, and was re-admitted soon afterwards. He then suffered from severe pain of a paroxysmal character in the epigastrium, and extending to the sides and back, and of fixed pain in the left gluteal region. Pulse 84, and weak, but regular, and equal on the two sides; pupils normal and equal; cardiac impulse strong; in the scrobiculus cordis a loud bellows-murmur was heard, which was synchronous with the pulsation of the abdominal aorta. Occasionally, a second murmur was likewise heard in this situation, diastolic in time, cooing in character, and resembling very closely a musical venous murmur. The former was faintly heard over the left costal border near the xiphoid cartilage, but evidently by transmission. The treatment consisted in iron given in a bitter infusion, aperients, and a small blister to the gluteal region.

Discharged, much relieved, July 31st, and again admitted August 14th. For two days after leaving hospital he had been entirely free from pain, but during the remainder of the period to the date of re-admittance he had suffered from excruciating pain in the left side of the abdomen, shooting down to the left hip and also to the left groin and knee, in the course of the ilio-sciatic and anterior crural nerves. This pain was paroxysmal, causing a "burning" sensation in the left groin and back, and was aggravated when he assumed the erect posture. A *bruit de soufflet* was heard beneath the ensiform cartilage, synchronous with the pulsation of the abdominal aorta; it was soft and lisping in character, traceable upwards to left of ensiform cartilage, where both sounds of the heart were likewise audible; here it was heard to follow the first sound, but at an appreciable interval of time, whilst it immediately preceded the second sound without any interval. After a voluntary movement of the patient's body the murmur was likewise audible at the apex of the heart, where it was fainter, but otherwise unaltered, and held the same relationship to the sounds. Its point of maximum intensity was at the tip of the ensiform cartilage; here it was constant, and when audible in all three situations, might be followed from below upwards and to the left in an uninterrupted line of about three inches, but with decreasing intensity from the ensiform



was independent of respiration. Above the level of the xyphoid cartilage the murmur was occasionally suppressed during one or two pulsations, and on two several occasions the most careful examination in the recumbent posture failed to detect it beyond the limits of the scrobiculus cordis. Occasionally, a second murmur was heard immediately succeeding the former, and therefore coincident with the systole of the aorta; it was musical in character, and strictly localized. Pressure upon the abdominal aorta at any point below the seat of murmur had the effect of diminishing the force of the principal, and of suppressing the second or after-murmur. The heart pulsated in its usual situation, and the area of precordial dulness, and the cardiac sounds, were normal.

To have two leeches to seat of pain in epigastrium.

August 23rd. No murmur audible anywhere in front, save in the scrobiculus cordis, where it is double; the latter element occasionally musical.

24th. The principal murmur is again audible over line from apex of heart to scrobiculus. Over the last dorsal and first lumbar spinous processes, and to an extent of two inches around this point, a loud blowing murmur, but unattended with impulse, is heard. This part is tender on pressure, and the seat of "shooting," "burning" pain. To have two leeches applied here.

On the 2nd of September the pain on the left side of the abdomen and back was excruciating; the patient implored us to relieve him, and one-twelfth of a grain of muriate of morphia in solution was injected subcutaneously in the former situation, with the effect of completely subduing pain for a period of twelve hours.

The following remarks, written under date of 3rd September, are copied from my note-book:

"The collateral evidence here is sufficiently strong to warrant the diagnosis of inferior thoracic, or thoracico-abdominal aneurism, even in the absence of tumor and perceptible impulse. No doubt exists in my mind as to the non-cardiac nature of the murmur heard over the left costal cartilages, and as to its iden-



nous with the pulsation of the abdominal aorta, though modified by distance from the seat of its origin.

“ The interval of time that must elapse between the systole of the left ventricle, and the manifestation of its effects, by impulse and murmur, in an aneurism of the inferior thoracic or superior abdominal aorta, though infinitesimal, is nevertheless strictly calculable from a knowledge of the distance between the points mentioned, and of the rate at which the blood-wave moves in the individual under examination.\* If the interval so determined be appended to the period of the first sound of the heart, and the abdominal murmur which marks it be supposed to be transferred or transmitted to the area within which the cardiac sounds are audible, it is manifest that the murmur will be heard in the precordium at the close of that interval, and that it must coincide with some one cardiac phenomenon posterior in time to the first sound.

“ Now, if that phenomenon happen to be the negative one of the short pause, and the terminal portion of it, we shall have a murmur, not belonging to the heart, audible in the precordium, and coinciding with that portion of the short pause immediately anterior to the second sound. Such a murmur I would designate as *prediastolic*.

“ In the case under consideration, if one hand of the observer be placed upon the abdominal aorta, and the other in the site of the apex-pulsation, whilst the ear is applied to the stethoscope placed below the ensiform cartilage, the asynchronism between the impulse of the heart upon the one hand, and the pulsation of the aorta and the murmur on the other, will be readily perceived.”

Murmurs of this precise rhythm, not located at the aortic orifice, are invariably exo-cardial.

The patient's condition underwent some improvement, and he was discharged in the early part of October. I now lost sight of

\* Dr. Handsell Griffiths (*British and Foreign Medico-Chirurgical Review*, October 1868) has, as the mean of the results obtained from 300 experiments with his hæmadromometer, estimated the velocity of the blood-wave in the carotids of the dog at 400 millimètres (16 English inches) per second.

was then coughing up unmixed florid blood in considerable quantity. Pulse 120, regular, and equal; pain was now referred chiefly to lower part of left side of chest, which was absolutely dull, and devoid of vocal thrill and respiratory sound; the intercostal spaces were here obliterated, but tender to pressure, and the hand placed on this portion of the chest perceived a general heaving movement.

From the angle of the scapula upwards respiration was normal but accompanied with râles; a faint, rough systolic\* murmur was audible at the ensiform cartilage; the heart pulsated above the level of the nipple, and inside the nipple-line; its action was strong, and its sounds loud, clear, and normal.

A blister was directed for the side, and acetate of lead in gr. v doses as a styptic. On the 18th of March he was again admitted into hospital, and on the 19th, the following note of his condition was taken: Pulse 120; left side absolutely dull at all points below level of second intercostal space, and devoid of respiratory and vocal phenomena; impulse faintly perceptible here; above level of third rib percussion-resonance is of a hollow tympanitic character; in front, this part of the chest yields no respiratory sound, but posteriorly it is the seat of bronchial respiration with fine crepitus; over posterior inferior portion of left side two sounds are heard, but no murmur; and at the scrobiculus cordis a murmur is audible only on making firm pressure with the stethoscope. The patient was sleepless, and expectorating viscid pneumonic sputa; the lips were dotted with the crusts of dried herpetic eruption. To have gr. iss of aqueous extract of opium at night.

On the 9th of April it was noted that the left infra-clavicular region, from the median to the axillary line, had continued to yield a hollow tympanitic sound. Respiration had been for some days remarkably variable; at one time it was loud and puerile, and accompanied by coarse muco-crepitus, and on the following day all but suppressed; these alternations were of frequent occurrence. The heart was now displaced upwards, and pulsated visibly and

\* *I.e.*, representing the *systole* of the left ventricle in a *distant* artery, in the sense previously explained.

near the margin of the sternum; these spaces, nevertheless, yielded a hollow sound on percussion. The entire left side was enlarged, and the inferior costal cartilages of that side projected considerably; there was likewise a prominence in the inferior axillary region, and the patient was troubled with a loud ringing cough. The upper portion of the sternum, with the corresponding extremities of both clavicles, projected sharply, but this projection had existed from childhood, and was due to the natural conformation of his chest. There was no unusual dulness in this situation, but at the acme of expiration an obscure heaving pulsation was perceived by the hand placed flat upon it; and over its lower and right portion, corresponding to the attachment of the second costal cartilage to the sternum, and strictly limited to a space of about two inches in diameter, a harsh murmur, prediastolic in rhythm, was heard. The cardiac sounds were loud and free from murmur. The epigastric hollow was obliterated; the liver projected considerably, but equally; but its surface was even; the organ was not displaced downwards, nor did the hepatic dulness extend above the level of the sixth rib. Expectoration copious and purulent, and occasionally mixed with a little blood.

29th. The secretion of urine has been greatly diminished since last report, amounting for the last fortnight to not more than eight ounces in the twenty-four hours; it is loaded with lithates. Pulse 120, weak, but regular; respiration 48; cough troublesome and ineffective; loud puerile respiration for two inches below the left clavicle. A faint murmur, synchronous with abdominal pulsation, existed at the ensiform cartilage, where the sounds of the heart are faintly audible, but from this point upwards to the site of cardiac pulsation they increase progressively in distinctness. On the preceding night, the patient had been attacked with aggravated dyspnoea, accompanied with severe pain in the epigastrium, shooting down to the left groin. To have a sedative of hydrocyanic acid and morphia at night.

On the 6th of May a loud bellows-murmur, nearly coincident with the radial pulse, was detected in the sitting posture along the lumbar spine, and for two inches to the left; in this situa-



tion there was severe and fixed pain, which was not aggravated by pressure or by percussion; prediastolic murmur, remarkably loud and harsh over sternal prominence, and somewhat to right, in both of which situations the hand can detect a heaving impulse; between the site of this murmur and that of cardiac impulse no abnormal sound was discoverable; neither did murmur exist in connexion with the cardiac sounds. The pulsation of the heart was violent, lifting up at each stroke the corresponding portion of the chest-wall, which yielded a hollow sound on percussion. No respiratory sound was audible in left infra-clavicular space; there was some fetor of the breath. To have compound decoction of scopolarium with nitrous spirit of ether; also an anodyne at night.

9th. During my visit to the ward at 12 o'clock he was suddenly attacked with the most distressing dyspnoea, which caused him to start into the sitting posture; respiration was very rapid and laboured, and the cervical veins became turgid, but the face remained pale. On inquiry I learned that in the course of the preceding fortnight he had had several such fits, but none of so aggravated a character as this; there was great anxiety, and the patient besought me not to leave him. The dyspnoea continued, unmitigated by ethers and stimulants; the patient became gradually weaker, and died of exhaustion at 2 o'clock. On the evening of that day a *post mortem* examination of the body was made by the resident pupils, Messrs. Furlong and Kelly, under circumstances of much difficulty. To these gentlemen, and to Mr. Petit, I am indebted for the opportunity of exhibiting the morbid specimen to the Society,\* and of clearing up some doubts that arose in my mind in the course of the case.

The haste with which the examination was made will account for some injuries sustained by the viscera during removal.

On removing the sternum a good deal of serum escaped; whence this proceeded it is difficult now to say, but it probably came from the right pleura. The great veins at the root of the neck, and the cavæ, were distended with blood; the heart lay across the chest below the left clavicle, its apex to the left and

\* A report of this case was laid before the Medical Society, Wednesday, May 26th, 1869.



its base to the right side; it occupied the great pulmonary fissure, displacing upwards, and pressing into the cone of the pleura, the superior lobe of the left lung, the base of which overlapped and in great measure concealed it from view; it was rather below the average size of the adult heart, and was perfectly sound in its walls and in its valves. The arch of the aorta was free from disease, but the curve at the junction of its ascending and transverse portions was greatly exaggerated, owing to the displacement of the heart upwards. The superior lobe of the left lung was universally and firmly attached to the parietal pleura, and much reduced in volume by compression between the displaced heart and the superior osseous boundaries of the chest, but it was otherwise free from disease. The inferior lobe of the lung was entirely obliterated as such; it was converted into a thin lamina spread out upon, and firmly attached to, the surface of a vast, solid tumor which occupied nearly the entire of the left side of the chest. This tumor was found to be an aneurism, communicating with the thoracic aorta immediately above the diaphragm by an oblong opening about two inches by one inch in diameter, and engaging one-half the circumference of the vessel; the edges of this opening were rounded off, and expanded into a vast aneurismal pouch which extended vertically from the second rib to the left kidney, displacing the lung and heart upwards, the diaphragm downwards, the liver forwards, and the ribs and costal cartilages outwards. It was firmly attached to the chest-wall, from which it was removed with great difficulty, and inseparably so to the diaphragm beneath the external arched ligament; thence it had descended behind the left kidney, and was attached to the upper extremity of that organ. The sac of the aneurism, which was formed by condensation of the surrounding tissues, was lined on three aspects by dense laminated fibrin; viz., inferiorly, externally, and posteriorly; but superiorly, and also internally, it was composed of little more than the pleura; whilst anteriorly, as already stated, it was strengthened by the condensed and adherent lung-tissue. The sac contained a great quantity of dark clotted blood, and in its upper part, a mass of shreddy fibrin mixed with coagulum.

There was no opening from the sac save that which communicated with the aorta.

The bodies of the sixth, seventh, and eighth dorsal vertebræ were deeply and extensively eroded ; the seventh had been almost entirely destroyed. The corresponding intervertebral fibro-cartilages were scarcely affected. The œsophagus, trachea, and bronchi had escaped pressure. The right lung was healthy, as were likewise the liver, spleen, and kidneys ; but the left renal artery was pressed on and nearly obliterated by the tumor.\*

In cases of this kind little can be done curatively ; indeed nothing beyond mitigating the sufferings of the unhappy patient and postponing the final event, whether from expansion or rupture of the sac, by means of regimen and appropriate medicine.† The interest connected with such cases, therefore, has reference mainly to diagnosis.

A short *résumé* of this case may suggest profitable reflection.

A young man of intemperate habits strained himself in lifting a weight ; shortly afterwards he complained of pain in the back ; two years subsequently he presented himself, and was then suffering from acute pain in the lower part of the back, which extended to the left side of the chest, the left hip, and groin. This pain was intermittent and paroxysmal, and described as being of a "burning," "boring," character, and aggravated by the patient's assuming the erect posture. A murmur was heard at the ensiform cartilage, posterior in time to the first sound of the heart, anterior to the second sound, and synchronous with the pulsation of the abdominal aorta ; a second murmur of a musical character, and coinciding with the systole of the aorta, was also heard occasionally in this situation ; the former of these murmurs was modified, and the latter extinguished, by pressure upon the abdominal

\* *Vide* Fig. LXXII.

† Since the publication of this paper in *The Dublin Quarterly Journal* for August, 1869, I have been favoured with a copy of a very interesting and valuable pamphlet on the treatment of aneurism by means of iodide of potassium in large doses, by Dr. George W. Balfour, of Edinburgh. Since receiving the pamphlet, for which I beg to express my obligations to Dr. Balfour, I have made partial trial of his treatment, and feel bound, so far, to report favourably of it. In the only case of the kind in which I have since had an opportunity of administering the iodide, namely, one of abdominal aneurism, the excruciating pain promptly ceased after its exhibition.

aorta below the seat of their origin; a single bellows-murmur was audible over, and in the vicinity of, the lower dorsal vertebræ, which were the seat of pain and tenderness, and the centre whence pain radiated into the chest and abdomen; no impulse was discoverable in the epigastrium, and no murmur below the ensiform cartilage. The action and sounds of the heart were normal; there was no febrile excitement; vital functions performed satisfactorily, and general nutrition unimpaired. Five months subsequently, the left side of the chest was found enlarged, and dull on percussion, from the base to the angle of the scapula; to the same extent respiration and vocal vibration were abolished; the heart pulsed above and inside the left nipple; respiratory sound was heard superiorly, and here percussion sound was hollow and tympanitic.

The patient was pale, expectorating pure florid blood, but free from hectic symptoms; his pulse was quick and feeble.

Three weeks later, the liver was found pushed forwards into the epigastrium, but not increased in volume; the heart was displaced upwards beneath the clavicle, the ribs were everted, and the inferior costal spaces obliterated; a heaving pulsation, accompanied with a double sound, was at the same time perceptible over the inferior posterior portion of the corresponding side. The murmur at the ensiform cartilage persisted, but was then much less distinct; a bellows-murmur, slightly posterior in time to the radial pulse, was heard along the lumbar spine; renal secretion was partially suppressed; the patient became gradually weaker; paroxysmal dyspnoea supervened, and in one of these fits he expired.

The foregoing sketch, viewed in its entirety, no doubt leads necessarily to the diagnosis of aneurism of the inferior thoracic aorta. But it is no less manifest that certain portions of it, not interpreted by the light of the remainder, might seem to warrant a different conclusion. Thus, for example, if the patient were seen by a medical man for the first time when the left side of the chest was enlarged, dull on percussion, and devoid of respiratory and vocal phenomena throughout the greatest part of its extent, whilst superiorly there was persistent modified resonance with variable respiratory sound, the latter being, as repeatedly hap-



FIG. LXXII.



- 1-2 Superior lobe of left lung overlapping and attached to the heart. (1, Lung tissue in section.)
- 3 Heart (pericardium removed) occupying pulmonary fissure, and flattened by pressure.
- 4 Inferior lobe of lung flattened out and incorporated with aneurismal sac (seen in section.)
- 5 Aneurismal sac, torn in process of removal.
- 6 Jagged and coloured fibrin occupying aneurism.
- 7 External surface of sac formed by thickened pleura.
- 8 Empty portion of sac.
- 9 Diaphragm expanded upon and attached to sac.
- 10 Opening in aorta (two probes introduced into the vessel).
- 11 Everted portions of torn sac.
- 12 Left kidney.



pened, loud, and accompanied with coarse muco-crepitus, amounting almost to *gargouillement*, one day, and on the next entirely suppressed; the patient expectorating fetid, blood-stained pus, and the liver and heart displaced, the diagnosis of primary disease of the lung, probably malignant, with copious secondary liquid effusion into the pleura, would, not improbably, have been made.

The annexed engraving (Fig LXXII.) exhibits the aneurism in the foregoing case, and also the relative position and general appearance of the parts related to it.

**CASE CXXXVIII.**—*History of Intemperance and Injury of Chest by Strain; Cough, Orthopnœa, Stridor, and Paroxysmal Dyspnœa from Spasm of the Glottis; Imperfect Respiratory Movement on both Sides of the Chest, with almost complete Suppression of Respiratory Sound on the Left Side; Absence of most of the Collateral Symptoms, and Total Absence of the Proper Signs of Aneurism; Laryngotomy, followed by Complete Relief from all the Urgent Symptoms; Death more than a Month subsequently from Pneumonia. Autopsy: Aneurism arising from the Posterior Wall of the Transverse Portion of the Arch of the Aorta at its Left Extremity, and Pressing upon the Trachea; Necrosis of the Larynx, and Double Pneumonia.*

Thomas B., aged thirty-three years, by occupation a packer in the egg market, and very intemperate, entered the Mater Misericordiæ Hospital on the 13th of March, 1871. Three weeks previously, whilst carrying a heavy hamper of eggs, he was suddenly seized with pain in his back, and difficulty of breathing. A week later he entirely lost his breath after ascending a flight of stairs, but felt no pain, and at the end of another week a loud, wheezing noise accompanied his breathing.

Latterly, both the respiratory distress and the stridor had become more troublesome, and for the three weeks immediately preceding admittance he had had cough. He had never spat blood.

When admitted, he was pale, with a remarkably anxious ex-

pression of face; respiration was embarrassed; the patient was unable to lie down, and a faint stridor accompanied inspiration. There was cough, with expectoration of mucus, which, as he declared, frequently "gathered at the root of his neck," causing increased distress of breathing, and was brought up with much difficulty by coughing.

No venous engorgement or dysphagia. Pulse 84, small, regular, and equal on the opposite sides. Pupils normal and equal. Voice somewhat husky. Precordial dulness, site of apex-pulsation, and sounds of heart normal, but impulse feeble. No abnormal impulse, and no murmur anywhere discoverable. Respiratory movements were imperfect on both sides of the chest, but in a greater degree on the left side than on the right; respiration was likewise feeble, and masked by loud stridor on both sides. The slightest movement of the patient's body, such as that required in drawing on his clothes, was capable of inducing a paroxysm of the most urgent dyspnoea, accompanied by loud stridor. On examination with the laryngoscope, which was made with much difficulty owing to the distress which it occasioned the patient, the vocal chords were seen to move equally, and no tumor, cedema, or other cause of obstruction was visible.

The paroxysms of dyspnoea became gradually more severe and more frequent; they likewise now occurred irrespectively of effort on the part of the patient, but were in some degree alleviated by large doses (grs. xx) of bromide of ammonium.

On the 10th of April he had several paroxysms accompanied by loud stridor, each lasting about an hour, during which he partially lost consciousness. He experienced some relief from ℥xxx of wine of ipecacuanha, with ℥x of tinct. of cannabis Indica, given during the paroxysms.

On the 13th of April, the patient being nearly asphyxiated in a paroxysm of unwonted severity and duration, laryngotomy was proposed, but the poor man requested it should be postponed till the following day.

On the 14th, however, urgent dyspnoea with loud and shrill stridor having continued with unremitting intensity through the preceding twenty-four hours, laryngotomy was performed by my colleagues, Messrs. Cruise and Hayes, with much difficulty, owing

to the extreme restlessness of the patient. The cricoid cartilage and the first ring of the trachea were divided. Considerable relief followed the operation, as the patient lay down immediately afterwards and obtained some tranquil sleep; he likewise took some wine and water, which previously he had been unable to swallow, owing to the dyspnoea which the attempt occasioned.

16th. He had passed a good night, having slept several hours after a draught containing grs. xxv of chloral hydrate; respiration tranquil, and when the tube was closed with the finger, he could breathe freely through the glottis; he was likewise able to swallow liquids without difficulty. Pulse full and regular; slight bronchial râle; respiratory sound was loud and harsh on the right side, and almost absent on the left.

17th. Took chloral draught, and slept through the night after 10 o'clock; feels much better, and desires to know when the tube might be removed. After this date, tumefaction and ulceration appeared about the wound, and were much benefited by warm stuping, and dressing with oxide of zinc ointment, the edges of the wound having been protected from the pressure of the tube by means of strapping with adhesive plaster.

On the 29th he could breathe freely through the larynx, and speak in an audible voice; but he could not expectorate save through the tube. The expectoration, which had been previously copious and muco-purulent, was then much reduced in quantity. He could swallow solids with facility, and sleep in the recumbent posture. The bowels and kidneys were acting naturally. Respiratory murmur, which for two days after the operation had been all but inaudible in the left lung, was now equally distinct on both sides; the action and sounds of the heart were normal, the radial pulses were regular and equal, no abnormal dulness or pulsation was anywhere perceptible, and no pain or tenderness was referred to any point of the chest.

Shortly after the last mentioned date, the patient began to expectorate pus in large quantity. About the middle of June the sputa became pneumonic; pain was felt over the base of the right lung, and here the physical signs of pleuro-pneumonia were detected. Despite treatment, and with intervals of slight

improvement, the man's condition gradually became worse; he died, exhausted, on the 23rd of June.

On examination of the body, serum, to the amount of eight ounces, was found in the pericardium, but no evidence of pericarditis existed. A large "milk-spot," raised above the surface, and covered with epithelium, was found on the anterior surface of the right ventricle; another, of smaller size, existed on the right side of the apex. The heart was healthy in structure but slightly enlarged; it weighed eleven ounces. The valves were all in a normal condition. There was a good deal of decolorized fibrin in the right auricle, and in both ventricles, whence it extended into the aorta and pulmonary artery. The lower lobe of the right lung was covered with false membrane, and in the third stage of pneumonia; the inferior lobe of the left lung was congested and solidified.

The lining membrane of the aorta, throughout the arch and the descending portion, was of a deep crimson tint, and the left extremity of the transverse portion of the arch was dilated, and mottled with specks of atheroma. In the posterior wall of the vessel, directly behind the left subclavian artery, and about three lines from the orifice of that vessel, a circular aperture, three-fourths of an inch in diameter, existed, leading into an aneurism of the size of a bantam's egg. The edges of this aperture were even, but fissured by scaling of the lining membrane.

The aneurism projected into the antero-left wall of the trachea, which was here reduced to one-half its normal diameter, the wall of the trachea being much attenuated, and its lining membrane congested, at the point pressed upon. The sac was nearly empty, a few flakes of fibrin only adhering to its posterior wall; it was flaccid, and rugose on the internal surface. The left recurrent laryngeal nerve took its normal course, and both it and the œsophagus had entirely escaped pressure.

The cricoid cartilage was completely and universally necrosed; it fell to pieces, as small, dark, and gritty particles, during removal. The lower edge of the thyroid cartilage was likewise in a state of necrosis.

In this case, aneurism had been diagnosed from the occurrence of paroxysms of laryngeal dyspnoea, unaccompanied by disease of



the larynx, and preceded by a definite history of strain and injury within the chest. The precise seat of the disease had been likewise approximately determined, mainly from the feebleness of respiratory sound in the left lung. The principal interest connected with the case, however, arises from the relief obtained by laryngotomy.

\*

CASE CXXXIX.—*Severe Neuralgic Pains, with Diaphragmatic Pleurisy ; Harsh Dry Cough ; no Special Symptom of Aneurism, and no Sign, except Sharp Second Sound in the Ascending Portion of the Arch of the Aorta ; Sudden Death. Autopsy : Hæmorrhage into the Pericardium by Rupture of a Large Dissecting Aneurism of the Aorta, engaging the entire of the Ascending and Transverse Portions of the Arch ; Fatty Accumulation upon the Heart, and almost Total Absorption of its Muscular Substance ; Valves not Affected.*

Mr. John B., aged sixty-two years, a man of great ability, and an architect by profession, had been, for more than three years, suffering from wandering pains, apparently rheumatic, in the muscles of the chest, back, and shoulders. On one occasion these pains were localized in the diaphragm, and associated with the signs of diaphragmatic pleurisy on the right side. During the whole of this period he had been under my care ; and a few months before his death, whilst suffering from pains referable to the diaphragm, he consulted Dr. Stokes. Neither of us detected any evidence of aneurism. Having repeatedly and carefully examined his chest, I can positively affirm that, with the exception of the neuralgic pains just mentioned, no symptoms suggestive of aneurism existed. Mr. B. was a man of very active habits, exceedingly nervous, and disposed to exaggerate his symptoms. He recovered from his last attack, that of diaphragmatic pleurisy, after a protracted illness, and resumed his professional duties ; but, as he informed me, he was no longer equal to his wonted activity, and fatigue of any kind told sensibly upon him. He continued, however, at his professional avocations, and was able to travel to distant parts of the country. A year later he called on me again, and complained of neuralgia

of the face and arms, but there was nothing in his condition to attract special notice. I treated him for neuralgia, and he seemed to have recovered his usual health.

He visited me, for the last time, on the 10th of November, 1871, to seek relief from neuralgia of the most aggravated character. He described the pains as shooting over the face, scalp, and neck, down the right arm, and around the right side of the chest; he pointed to one spot, midway between the right nipple and the sternum, as the seat of fixed pain. The pulse was 84, rather weak, but regular and equal; appetite good, and all the functions regularly and effectively performed. The pain usually ceased at night, and his sleep was unbroken and refreshing. The pupils were normal; he was not subject to vertigo or faintings, and had never spat blood; there was no arcus senilis, or oedema. He informed me that he had a harsh dry cough, which, however, I had not the advantage of hearing.

I made an examination of his chest, directing my attention only to the precordium and the seat of alleged pain to the right of the sternum. The action of the heart was quite regular, but the first sound was faint, whilst the second sound was remarkably sharp, loud, and abrupt at the base of the heart, and, in a still greater degree, at the right margin of the sternum, on a level with the second and third interspaces.

I made light of his case, and was about to prescribe for the neuralgia, which was the only symptom that seemed to demand attention. He was a very intellectual man, and both of us being interested in the subject of a passing conversation of the previous moment, I asked him a question in reference to it, whilst proceeding to write a prescription. He answered rather excitedly, as was his habit; I heard a rustling movement, and, turning round, I saw him fall off the chair towards the left side, and upon his face. It immediately occurred to me that he had been subject to epileptic seizures which he had hitherto concealed. I lifted him up, and at once saw that the matter was more serious. His neck became turgid, the pulse ceased, and he made a few gasping respiratory efforts. I turned him over on his back, having hastily summoned the servant from the adjoining hall; a dark shadow passed slowly over his face from below, manifestly

the result of reflux venous engorgement from the right side of the heart; and life had ceased. The whole period occupied by the occurrence just described did not exceed three minutes.

At the instance of the City Coroner, Surgeon N. C. Whyte, and with the assistance of my friend, Dr. E. W. Collins, I made a careful examination of the chest two days after death. The anterior mediastinum was occupied by large masses of yellow adeps; the pericardium was distended with serum and blood-clot, to the amount of a pound and a-half. The heart was somewhat enlarged, covered with fat, and remarkably soft and flabby. The aorta was greatly dilated immediately above the orifice, and the internal and middle coats, the latter of which was highly atheromatous, had given way, the blood insinuating itself between the middle and the external coat. The aorta had gradually expanded, from the sinuses of Valsalva upwards, into an aneurism of the size and shape of a pine-apple, capable of containing at least twelve ounces of blood, and engaging the entire circumference of the vessel as far as the descending portion of the arch. On the right side of this enlargement, half an inch above the valves, a rent was seen by which the blood found in the pericardium had escaped. This led upwards into a sinuous passage between the external and middle coats of the vessel, which extended over a considerable portion of its circumference, and ended in an irregular opening into the aorta nearly on a level with the reflection of the pericardium.

The internal surface of the sac, which contained some loose blood-clots, was rugose, highly atheromatous, and fissured in several places. The three primary branches of the aorta were strictly normal in diameter, and free, as was likewise that vessel itself, beyond the transverse portion of the arch. The trachea and the œsophagus had entirely escaped pressure. The left ventricle was dilated and hypertrophied, the outer two-thirds of the substance of its walls consisting of fat, which had accumulated on the surface of the heart and grown inwards, displacing and causing atrophy of the muscular substance.\*

The internal portion of the ventricular wall, seen in section, presented the normal appearance to the naked eye; its fibres

\* *Vide* Fig. XXXIV., p. 608.



exhibited under the microscope the ordinary transverse striæ.\* The fat cells were large, and partially collapsed, through the action of sulphuric ether in which the section examined had been previously soaked.†

The right ventricle was in a more advanced stage of fatty substitution; the entire thickness of its wall, except a film on the internal surface not thicker than a threepenny piece, having been replaced by fat. The right auricle was laden with fat, very

FIG. LXXIII.



Aneurism of arch of aorta and fatty heart. Mr. John B.

\* *Vide* Fig. XXXVI., p. 609.

† *Vide* Fig. XXXV., p. 609.



little of its muscular substance remaining. The valves were all healthy and competent. The most noteworthy feature in this case is the latency of a twofold disease of so formidable a character as aneurism of the aorta with fatty transformation of the heart. With the exception of harsh and dry cough, which I had not heard, and migratory neuralgic pains, localized, however, at one point of the chest, there was absolutely no symptom suggestive of aneurism, and none which would have directed special attention to the thorax as the seat of organic disease of any kind. The dull character of the first sound, and the sharp ringing quality of the second sound, excited a passing suspicion of weak heart and atheroma of the aorta; but, in the absence of more definite evidence, neither aneurism nor fatty disease of the heart was contemplated. Complete and careful examination of the chest was, therefore, not made on the occasion of the patient's last visit to me.

The annexed engraving (Fig. LXXIII.), from a drawing by Mr. Burnside, shows the appearance presented by the heart and the aneurism after dissection. The sac had been laid open; the uneven condition of its internal surface is represented. The openings of the three primary branches of the arch are indicated by bristles, and the continuation of the aorta, by the passage of a piece of gum elastic bougie.

CASE CXL.—*Aphonia, Stridor, Orthopnœa, and Paroxysmal Dyspnœa; Suppression of Left Radial Pulse, and Contraction of Left Pupil, with Ptosis; Subclavicular Dulness, and Feeble Respiration on Left Side; Copious Expectoration of Pus; Death by Asphyxia. Autopsy: Emphysema of both Lungs, and Pneumonia of Right Lung; Deposit of Fat upon Heart; Aneurism of Transverse portion of Arch of Aorta pressing upon Left Subclavian, displacing and stretching the Œsophagus, Right Pneumogastric and Left Recurrent Nerves; Larynx Healthy.*

Mrs. L. I., aged thirty-one years, entered the Mater Misericordiæ Hospital on the 2nd of January, 1865. She had enjoyed

good health till the early part of the preceding month, when, after exposure to cold, she was attacked with cough, and entirely lost her voice. For these symptoms she had been under treatment in one of the city dispensaries, and, deriving very little benefit therefrom, she sought relief in hospital.

Her appearance indicated great suffering; she was thin and haggard, and her face was pallid, with a tinge of lividity. There was complete aphonia, with orthopnoea, and loud stridulous breathing, audible at a considerable distance from the patient. The slightest movement of her body, and even the exposure of her chest for the purpose of examination, was immediately followed by a paroxysm of coughing, accompanied by copious expectoration of frothy mucus. The right radial pulse was 108, and weak, but regular; whilst in the left brachial and radial arteries no pulsation could be detected. With a suspicion of intra-thoracic tumor before my mind, I made a careful examination of the chest, which, owing to the intensity and general diffusion of bronchial stridor, I found it impossible to carry out to my entire satisfaction. The only signs of a positive kind which could be detected were slight dulness under both clavicles, and comparatively feeble respiration in the left infra-clavicular space. On the day subsequent to that of admission, I tested the power of swallowing, and found it to be unaffected.

On the 7th, I observed a marked contraction of the left pupil, which continued in the shade. The larynx, examined with the laryngoscope, was found to be structurally healthy, and the vocal chords were seen to move normally in respiration. At my request, Dr. Hughes made a careful examination of the patient; he declared that, except contraction of the pupil, and suppression of the radial pulse (both on the left side), he could discover no evidence of intra-thoracic tumor. Dr. Hughes detected feeble pulsation in the left brachial artery.

On the 8th, partial ptosis likewise existed on the left side, the upper lid drooping to the level of the equator oculi. During the fits of spasmodic dyspnoea and cough which were consequent upon the slightest movement or exposure of the patient, pus was expectorated in large quantity, to the momentary relief of the patient.

The pulse became weaker, the respiration more rapid, and, notwithstanding that food was taken in moderate quantity, the patient sank very quickly. She died on the night of the 15th in a paroxysm of dyspnoea.

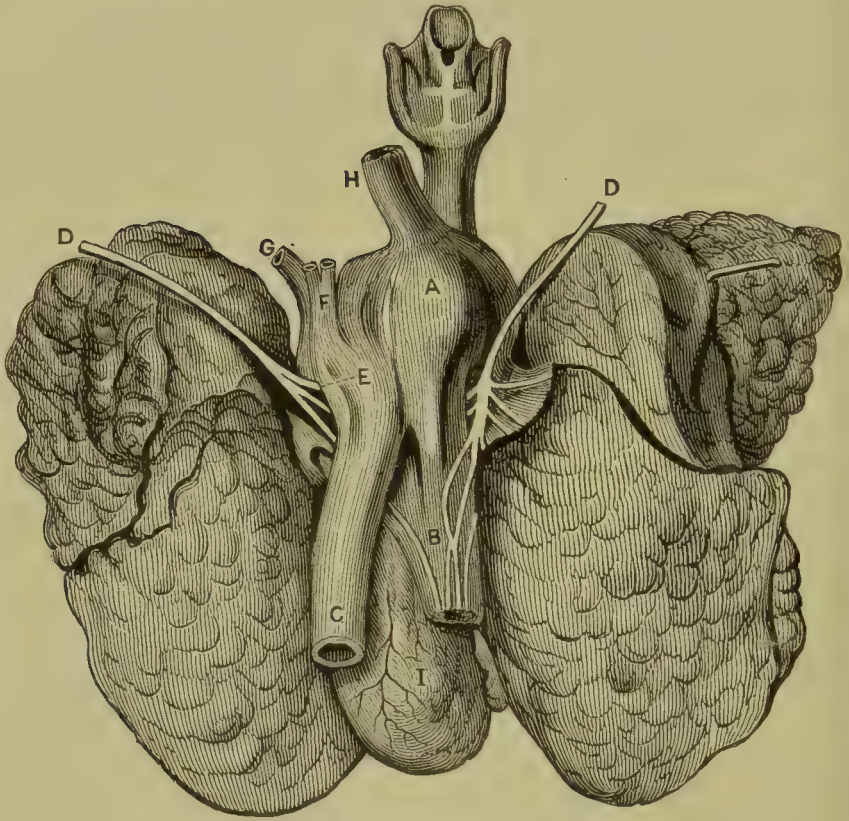
On examination of the body, both lungs were found to be emphysematous; the left being attached to the chest-wall by recently effused lymph and its base in the third stage of pneumonia. The larynx was healthy. There was a deposit of fat upon the surface of the heart, and the left ventricle contained some decolorized fibrin. The valves were all in a normal condition. An aneurism, of the size and figure of a large peach, had sprung from the transverse portion of the arch of the aorta, extending from the arteria innominata to the origin of the left subclavian, and pressing upon the latter vessel to the extent of an inch beyond its origin. It had arisen from the superior and posterior wall of the vessel, passed backwards to the vertebral column, by which it was indented, and thence to the right side behind the trachea, which had, however, escaped pressure. The œsophagus had been carried into the right pleural cavity by the advancing aneurism, upon the surface of which it was stretched, and firmly attached; it readily permitted the finger to pass through its canal at the seat of compression, and returned lower down to its normal position in the posterior mediastinum. The left carotid sprang from the sac, but was not diminished in calibre; the left subclavian was nearly obliterated as far as the origin of the vertebral artery. The direction of the arch of the aorta was so altered, that the branches arising from its transverse portion held an antero-posterior relation to one another. The right pneumogastric nerve was stretched upon the outer surface of the sac, whilst the left recurrent, extended upon and inextricably involved in it, had been carried over to the right side of the spinal column. The vertebræ were not eroded.

The paroxysms of dyspnoea and stridor, which were manifestly of a compound character, both laryngeal and bronchial, were explained by the state of tension in which the left recurrent and the right pneumogastric nerves were found; excitement of any kind, by increasing the force of the heart's action, must have caused temporary expansion of the sac, and so increased the



tension of these nerves. The position of the tumor was such that the left vertebral ganglion, and the superior dorsal portion of the left sympathetic chain, could not have escaped being crushed by it. Hence, contraction of the left pupil, with ptosis.

FIG. LXXIV.



Aneurism of the transverse portion of the arch of the aorta. Mrs. L. I.

- A Posterior view of aneurism, with œsophagus attached to, and expanded upon, it.
- B Lower portion of œsophagus, with *plexus gulce*.
- C Descending thoracic aorta.
- DD Pneumogastric nerves.
- E Left recurrent laryngeal nerve at point of incorporation with aneurism.
- F Left subclavian artery, giving off vertebral, and pressed upon by aneurism.
- G Left carotid artery.
- H Cervical portion of œsophagus.
- I Heart seen from behind, and laden with fat.

The lungs, in an emphysematous condition, are shown on either side.



Absence of the physical signs of aneurism must be attributed to the masking of the tumor by emphysematous lungs. The absence of dysphagia, notwithstanding the displacement and flattening of the œsophagus by the sac, is worthy of special notice. I believe this was due to the want of counter-pressure, and of ulceration of the gullet (see remarks at p. 1102).

The annexed engraving, from a drawing by the late Mr. Connolly, represents a posterior view of the parts after dissection.\*

CASE CXLI.—*Strain of Back and Chest, followed by Pain and Syncope; Dysphonia, Cyanosis, and great Œdema of Upper Half of Body, with Depression of Temperature; Feeble Respiration in Apex of Right Lung, with Dulness, Impulse, Thrill, and Murmur in the same situation; Death by Coma. Autopsy: Œdema of Lungs; Aneurism of Ascending Aorta pressing upon Right Bronchus, Right Branch of Pulmonary Artery, and Descending Cava, and opening into the latter vessel, thus forming an Arterio-venous or Varicose Aneurism.*

Michael W., a mason, of intemperate habits, aged thirty-three years, was received into hospital on October 17th, 1865. His health had been failing for the last year, during which he had undergone many privations; five weeks prior to admittance, whilst lifting a heavy stone from the shoulders of a fellow-workman, he made a false step, and strained his back. He complained, at the moment, of acute pain in the dorsal region, and was compelled to relinquish work for some days. He then resumed his labour, and suffered no further inconvenience till a fortnight later, when he experienced a sudden faintness, not accompanied by pain, whilst walking in the street, and would have fallen had he not supported himself against some fixed object. After a few moments this feeling passed away, and he was able to proceed. About this time I saw him as an out-patient at the hospital; he seemed to be under the influence of drink, was hoarse, and had cough. I did not examine his chest on this occasion. Three weeks later he presented himself again, and, owing to the striking peculiarity of his appearance, was at once admitted. The face, neck, and chest were swollen and

\* Vide *Proceedings of the Pathological Society of Dublin*, vol. ii., part iii., new series.

deeply cyanosed, the face and neck being of a dark purple tint, as if stained with dilute ink. The eyes were prominent; the conjunctivæ injected with venous blood, and the pupils contracted. The neck was bloated, so that the sub-maxillary and supra-clavicular fossæ were obliterated. The cervical veins were distended, but devoid of pulsation or thrill. The superficial veins of the chest and arms were distended and tortuous, and the finger-ends were livid. The upper half of the body was cold, and the man complained of constant chill, whilst below the diaphragm the surface was of the normal colour and temperature; the feet were warm. Respiration 24; voice somewhat husky and muffled; cough, with mucous expectoration; pulse 114, small, but regular, and not visible; tongue moist and livid; bowels natural; no dysphagia.

The chest was abnormally resonant in front, but less so on the right side from the clavicle to the level of the nipple. Respiration was puerile on the left side; over the lower portion of the right side it was likewise puerile, but above the nipple in front, and the middle of the supra-venous fossa behind, it was feeble. On both sides mucous râles were audible. Precordial dulness was somewhat extended. The heart's action was regular; the impulse was strong, and unaccompanied by *frémissement*; the apex pulsated at the xyphoid cartilage. At the base of the heart two bellows-murmurs were audible. One of these, systolic in rhythm, harsh and loud, was transmitted upwards through the arch of the aorta, and into the carotid arteries; it was diffused over the entire chest anteriorly, but was louder on the right than on the left side, and had its point of maximum intensity at the third right costo-sternal junction, where the second murmur was not audible. It was likewise heard in the right infra-spinous fossa, and was there associated with systolic impulse. The second murmur was diastolic in rhythm, rather faint, and nearly masked by the former; it replaced the second cardiac sound, and was strictly confined to the lower portion of the sternum. A feeble impulse, at the acme of forced expiration, was perceptible beneath the right clavicle.

On the night of the 20th the man was delirious; and, on the following day, nine leeches were applied at the upper part of the

sternum; the leech-bites bled freely, with great relief to the patient, and it became necessary to arrest the hæmorrhage by means of lunar caustic.

On the 22nd, the œdema had increased, and the patient complained of great tightness in his neck; the impulse beneath the right clavicle was now accompanied by a feeble thrill. An active diuretic mixture, including large doses of acetate of potash, had been hitherto given.

On the 23rd, owing to the great increase of lividity, œdema, and venous tension, I deemed it necessary to take blood from the arm; eight ounces were drawn, with great relief to the patient. The superficial veins of the left forearm were inflamed and marked by red lines on the 24th; leeches and poultices were applied. Cyanosis of the upper half of the body was now extreme, whilst the lower half retained its natural appearance. I wished to have a photograph taken, but the patient's consent could not be obtained.

On the 27th, voice was nearly extinct; he complained of an overpowering sense of drowsiness, and died soon afterwards, rather suddenly, in a state of coma.

On examination of the body, the lungs were found to be very voluminous, and engorged with blood and serum; their specific gravity was not, however, much altered. The pericardium, which was completely enfolded by the lungs, was much thickened, and adherent to the heart throughout. The heart was of the normal size and consistence; the right auricle contained a mass of dark blood-clot; the valves were all healthy.

The superior cava, at its junction with the auricle, was flattened from before backwards, and its opposite walls were attached to one another, leaving a minute passage into the auricle at either side; one of these passages admitted the little finger, but through the other a probe only could be introduced.

The aorta was considerably dilated, and its lining membrane was dotted with minute points of atheroma. A large opening, of an ovoid figure, one inch and three quarters by one inch in diameter, extended across the posterior wall of the aorta, half an inch above the valves. The margins of this opening were smooth but irregular; it led into an aneurism as large as a middle-sized

apple. The walls of the sac were thick and smooth, but rugous; it contained some loose masses of fibrin and dark coagulum. The right branch of the pulmonary artery had been pressed upon, and all but closed, by the aneurism, which had likewise, by its advance forwards and outwards, compressed the descending vena cava, and, as above stated, nearly occluded that vessel, from its entrance into the auricle to a point half an inch below the junction of the vena azygos. The last mentioned vessel was greatly enlarged. The sac of the aneurism had communicated with the vena cava by three openings, one of which was as large as a threepenny piece, the others being much smaller. The edges of these openings were thin and jagged, and on the venous side each was bordered by a deep red line. The superior primary branch of the right bronchus had been likewise compressed by the aneurism. Hence, feeble respiration in the upper lobe of the corresponding lung. Dysphonia was most probably due to oedema of the glottis; but this cannot be positively affirmed, as permission could not be obtained to examine any part of the body except the chest.

The diagnosis of aneurism had been made from the following facts and circumstances: The commencement of illness with strain, followed by pain in the back, and faintness; the evidence of pressure upon the superior cava by a tumor, and the existence, at the point where such pressure must have been exercised, of percussion-dulness, feeble respiration, impulse, and thrill. The presence of impulse, also, in the right scapular region, whilst the apex of the heart pulsated at the xyphoid cartilage, proved at once that two distinct centres of pulsation existed, and that the superior of these two occupied such a position, and had acquired such dimensions, as to afford a rational explanation of the venous and bronchial obstruction above mentioned, and, finally, the existence of a harsh systolic murmur, audible in its greatest intensity at the second centre of impulse.

The absence of well marked impulse, positive dulness, and dysphagia, was explained by the position of the aneurism with relation to the lungs and œsophagus.\*

\* Vide *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., p. 4.



CASE CXLII.—*Hæmoptysis, Dyspnœa, Huskiness of Voice, Stridulous Breathing and Cough; Tumor, Yielding Double Impulse and Sound, beneath Right Sterno-Clavicular Articulation; Feebleness of Right Radial Pulse; Tracheal Respiration at Upper Portion of Sternum; Bronchial Respiration in Right Scapular Region, and Feeble Respiratory Sound at Apex of Left Lung; Great Increase of Tumor, and Dislocation of Clavicle; Neuralgic Pains, and subsequently Œdema of Right Shoulder and Arm, Right Side of Chest and Neck; Contraction of Right Pupil, with Ptosis, and Elevation of Temperature on Right Side; Partial Suppression of Respiration in Upper Portion of Left Lung; Increased Dyspnœa and Stridor; Death. Autopsy: Acute Miliary Tuberculosis of both Lungs; Heart Hypertrophied and Fatty; Large Fusiform Aneurism of Ascending and Transverse Portions of Arch of Aorta, pressing upon Trachea, Left Bronchus, and Descending Cava, Perforating Sternum and Ribs, and becoming Diffused beneath Pectoral Muscles; Sac containing detached Laminated Fibrin.*

Bernard B., aged fifty-four years, a labourer, of intemperate habits, admitted into hospital in February, 1875. Had bronchitis two years ago, and has spat blood. His voice became husky five weeks before admission, and a fortnight later, he noticed the swelling, to be described presently, on the front of his chest. The following was his state on admission: Voice somewhat husky, and respiration slightly stridulous (from below); there was occasional dry cough, of a harsh, laryngeal, and metallic character. Slight dyspnœa, with darting pains in the right mammary region passing round to the axilla. There was neither engorgement nor pulsation of the cervical veins, and no congestion of the face, or fulness at the root of the neck. Pulse 84, regular, and moderately full, but slightly weaker in the right arm than in the left. Pulsation of carotids equally distinct. Between the second and third costal cartilages of the right side, and close to the edge of the sternum, there was a soft tumor of a conical figure, dull on percussion, and tender to pressure; it was about two and a-half inches in diameter at the base, and projected about an inch and a-half above the level of the surrounding surface; it communicated to the hand a double shock, viz.:

an impulse of an expansile character, coinciding with that of the heart, and preceding slightly the pulse at the wrist; and a diastolic impulse of arrest. On auscultation it yielded two sounds closely resembling the normal sounds of the heart, the first, however, being somewhat less pronounced than the corresponding cardiac sound, and coinciding with the principal impulse, whilst the second was sharp and accentuated. There was no murmur. Dulness extended horizontally three inches to the left of the tumor; namely, to a vertical line touching the clavicle one inch beyond its sternal extremity; on the right side it extended as far as the middle third of the clavicle. The *manubrium sterni* was slightly prominent, and respiration was here tracheal. A few engorged cutaneous veins were visible below the tumor.

The pupils were normal and equal; respiration was slightly accelerated, and became stridulous after the effort of sitting up in bed; respiratory sounds were equal on the opposite sides in front, and, with the exception above mentioned, strictly normal; posteriorly, respiration was slightly bronchial in the right scapular region; no dysphagia.

After he had been a few days in hospital, he complained of fixed aching pain in the right shoulder, which subsequently extended up the right side of the neck, above the ear. This was relieved by laudanum fomentations. He was strictly confined to the horizontal posture; was allowed full diet, and four ounces of wine. Two leeches were applied below the tumor, where he complained of sharp pain for one day, and tincture of the perchloride of iron was given in doses of ℥xv thrice daily. A chamois pad was strapped down over the tumor, and a sleeping draught, consisting of grs. xv of chloral hydrate, with ℥x of Battley's sedative, in an ounce of water, was given every night.

March 22nd. The tumor has enlarged and advanced considerably, tilting forwards the sternal end of the right clavicle and the clavicular portion of sterno-mastoid muscle; it has become firmer. Pulse 100, regular, and now equal. (See subjoined tracings, Figs. LXXV. and LXXVI.) Pain in shoulder and neck less troublesome. Acetate of lead, in gr. v doses, thrice daily, was now substituted for the iron previously given. Iodide of potassium was subsequently given, and at a later period phos-

phorus (one-thirtieth grain, thrice daily) was made trial of for a week. The benefit derived was not appreciable, except from the iodide, which relieved his pain.

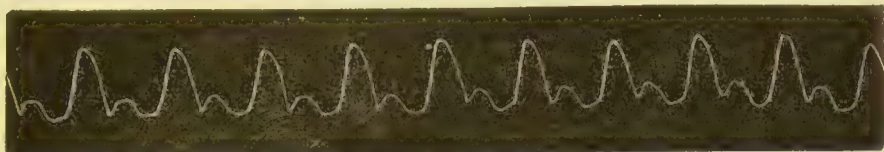


FIG. LXXV.

Aneurism of arch of aorta. Bernard B.  
Right radial; pulse 100. March 18th, 1875.

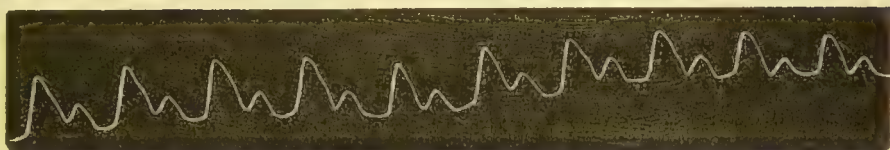


FIG. LXXVI.

Aneurism of arch of aorta. Bernard B.  
Left radial; pulse 100. March 18th, 1875.

April 7th. Respiration all but suppressed in the upper portion of the left lung. Tumor now projects more than two inches; it is elongate vertically, appearing behind the sternal end of the clavicle, which is dislocated upwards and forwards, and ascending nearly to the level of the cricoid cartilage. It measures at the base six inches by five, is yielding at all points, and, on the upper portion of the anterior surface, it presents a secondary prominence of a deep purple tint surrounded by enlarged cutaneous veins. In this situation the sac seems to consist of the integument only, and to be on the point of giving way. A double impulse, now very feeble, and a faint double sound unaccompanied by murmur, are still perceptible over the tumor. Pulse not detectable on the right side; in the left arm it is 108, irregular, and extremely feeble. Respiration 24, long-drawn and stridulous, both with inspiration and expiration. Contraction of the right pupil, with ptosis on the same side; œdema of the right arm, and of the right side of face and neck. Temperature in right axilla, 99° F., in left axilla, 98° F. Four ounces of blood were drawn by venesection, whilst the circulation was arrested in the right carotid by means of finger-pressure; the object of the venesection was, to prevent



increased tension of the sac whilst an attempt was being made to effect coagulation of its contents by arresting the circulation through it. This experiment, proving very irksome to the patient, was discontinued after half an hour, a period obviously insufficient for testing its utility. Ice was applied to the tumor with the twofold object of alleviating local tenderness and promoting coagulation in the sac.

8th. Patient was delirious last night; is slightly incoherent to-day; respiration loudly stridulous; ptosis and contraction of pupil, with congestion of conjunctiva, in right eye; temperature in the right external auditory canal, 97° F., in the left, 93.4° Galvano-puncture was now proposed with a view to effecting coagulation in the sac; but the patient, having been candidly told in reply to his inquiry, that the prospect of success from the experiment was not encouraging, declined to submit to it.

9th. Death occurred by slow asphyxia at 9.30 a.m. to-day, respiration having been greatly embarrassed, and attended with loud stridor for some time previously.

The body was examined on the following day. The lungs were studded throughout with disseminated miliary tubercle; at the apex of the right lung there was some thickening of the pleura, with old adhesion, but, with these exceptions, no trace of disease existed in either lung. The heart was somewhat enlarged, and remarkably fatty on the surface; the right ventricle was dilated, its wall was thinned, and in the outer three-fourths of its thickness, composed of fat; the left ventricle was slightly dilated and somewhat thickened; the valves were all healthy. The aortic orifice was normal, and its valves were competent. The ascending portion of the vessel was dilated to the size of a man's wrist; its walls were thick and rigid; and, at the junction of the ascending with the transverse portion of the arch, an aneurism sprang from it, which involved the entire transverse portion as far as the origin of the left subclavian artery. It was of the consecutive false kind, had perforated the manubrium of the sternum, in which an opening as large as a half-crown piece, with thin, irregular, and spiculated edges existed, and had dislocated the clavicle from the sternum. A circumscribed false aneurism, of the dimensions previously stated, had been formed in front of the chest beneath the pectoral muscles and aponeurosis, the



covering of which had been reduced at one point to the integument. The entire of the first portion of the sternum, the inner extremity of the right clavicle to the extent of an inch and a-half, and about an equal length of the first and second costal cartilages, which were completely ossified, projected into the aneurism, and were denuded of periosteum. The extremity of the clavicle, and that of the first rib with a piece of the sternum attached, were quite free within the sac. The lining membrane of the ascending portion of the aorta was of a deep crimson tint, and studded with minute yellow specks of atheroma. The sac was occupied by a firm and laminated shell of fibrin half an inch thick, which formed at its dome a *cul de sac*. This shell had been detached from the sac throughout, manifestly by the entrance of blood at some point of its circumference, and a false aneurism had been subsequently formed by rupture of the sac at its weakest point, under the pressure of the blood so infiltrated. The cavity of the fibrinous shell was smooth, and was occupied by soft loose coagula. The origin of the *arteria innominata* and left carotid were involved in the aneurism. The superior vena cava was stretched upon, and incorporated with, the exterior of the sac on the right side; it was greatly elongated, and its wall was, at two points, reduced to the lining membrane by the pressure of the sac. The left wall of the trachea, immediately above its bifurcation, had been pressed inwards by the aneurism, which formed a prominence within it; the secondary branch of the left bronchus supplying the upper lobe of the lung, had been likewise compressed, and was greatly reduced in calibre. The pneumogastric and recurrent laryngeal nerves of both sides were much thickened. The branches of the superficial cardiac plexus were in a similar condition. The mucous lining of the larynx was highly oedematous, the rima being nearly closed by the surrounding tumefaction. The bronchial glands were enlarged, and of a deep black colour.

In addition to the nine cases given in detail, including Case 69 (p. 759), in each of which a *post mortem* examination of the body was made, I have had under observation, but without the advantage of autopsy, seven other cases of thoracic aneurism. I

propose summarising these cases very briefly; and, for sake of uniformity, I shall do so with special reference to the several heads given in Tables XVI. and XVII.

CASE CXLIH.—William C., aged fifty-four years, a car-driver, of intemperate habits, admitted March 11th, 1868. Seven months ailing. General nutrition good. No history of strain or injury. Aneurism of the transverse portion of the arch, yielding a single and systolic impulse and two sounds, but no murmur. The direction of growth was forward, and there was no valvular disease or enlargement of the heart.

CASE CXLIV.—William R., a porter, aged fifty years, and temperate, admitted March 12th, 1868. Had been accustomed to lifting and carrying heavy weights. General nutrition good. Ailing three years. Aneurism of arch of aorta at junction of ascending and transverse portions, and projecting three inches above the surface at the upper right margin of the sternum. It yielded a double impulse and a double sound, but no murmur. No valvular or other cardiac lesion discoverable. Paroxysmal dyspnœa, with loud stridulous respiration, and harsh ringing cough. Relief of dyspnœa and cough from gr. x doses of the bromide of ammonium, and of pain in tumor from repeated application of ice.

CASE CXLV.—Benjamin W., a porter, had served three years in the army, temperate, and well nourished, accustomed to lift heavy weights. Ten months ailing. Aneurism of descending portion of arch, pressing upon left bronchus. No aneurismal impulse, sound, or murmur. Suppression of breathing in left lung; hæmoptysis; stridulous respiration; metallic cough, and recurrent paroxysmal dyspnœa with loss of consciousness. Death by asphyxia. Examination of body not permitted. Copious discharge of blood from the mouth and nostrils after death.

CASE CXLVI.—Mrs. C., aged thirty-five years, a farmer's wife, admitted May 11th, 1872. Illness commenced suddenly and without assignable cause four months previously, with sharp

pain in left axilla, which was followed by numbness of left arm, of a week's duration. Aneurism engaging left extremity of transverse portion of arch of aorta, projecting on left side of chest, from second to fourth intercostal space, close to sternum, and yielding a double impulse and double sound, with systolic murmur. Sharp shooting pains in left side of chest, left shoulder and arm, as far as elbow. Feeble respiration in left lung, and elevation of temperature, with local perspiration on left side. Double sound, without murmur, in left scapular region. Postponement of radial pulse. Mitral inadequacy. Pain relieved by leeching, and great benefit from ℞ doses of tinct. of the perchloride of iron.

CASE CXLVII.—John F., a farmer of intemperate habits, aged forty-two years, was received into hospital, July 13th, 1872. Five years previously he was thrown from a great height, and his chest came in collision with a stake. An aneurism of ascending aorta, projecting at right sterno-clavicular articulation, and yielding a systolic impulse and double murmur. Inadequacy of the aortic valves (relative?) and hypertrophy and dilatation of the left ventricle; severe shooting pain in right side of chest and neck, and right arm, relieved by hypodermic injection of morphia; much benefit from ℞ doses of tincture of the perchloride of iron.

CASE CXLVIII.—Mr. William B., an accountant, aged forty-five years, temperate, was brought to me by Dr. Brady, in June, 1873; nutrition satisfactory; no definite history of commencement of illness. Aneurism of ascending aorta, touching anterior wall of chest at right margin of sternum and level of third costal cartilage, within a space equal to a crown-piece in diameter, and yielding a systolic impulse and a double sound, without murmur. Great venous turgescence of face and neck. Paroxysmal dyspnoea with stridor, and increased venous distention produced by the slightest exertion; hæmoptysis. Forward growth of tumor. No valvular disease, or enlargement of heart. I again saw this gentleman with Dr. Hudson, on the 7th of June, 1875. He had had, in the interim, several attacks

of paroxysmal dyspnœa, accompanied by stridor, tumultuous action of the heart, and failing pulse. He had also repeatedly spat blood. The superficial veins of the chest and epigastrium were now enlarged and varicose. The jugular veins were likewise distended, and the face somewhat congested, but cyanosis had disappeared. Respiratory movement was defective on both sides of the chest, and respiratory sound less strong on the right than on the left side; there was persistent dyspnœa, and respiration was accompanied by stridor. The voice was slightly husky. Percussion-sound was now clear on both sides of the chest, and an impulse, with a double sound, was perceptible in the right interscapular space, and at a corresponding point in front. The lower half of the body was quite free from congestion. To inhale  $\text{M}_\text{v}$  of the nitrite of amyl for relief of paroxysmal dyspnœa, and have counter-irritation kept up on the right side of the chest.\*

CASE CXLIX.—George D., a labourer, aged forty-four years, of temperate habits, was admitted into the Mater Misericordiæ Hospital in December, 1873, and again on the 9th of January,

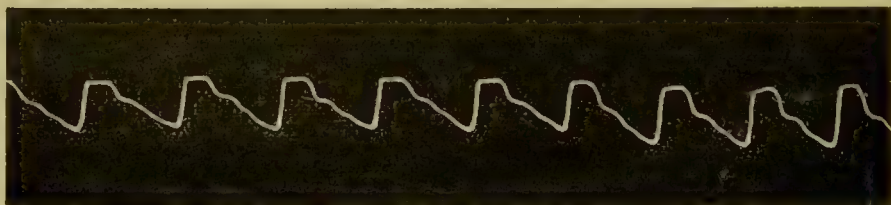


FIG. LXXVII.

Aneurism of arch of aorta. George D.

Pulse 96. January, 1874.

1874, under my colleague, Dr. Hughes, by whose kind permission I was enabled to examine the patient and make notes of the case. Illness commenced without definite cause three years previously. Pains in left side of chest, between scapulæ, and extending up the neck; fulness of left cervical veins; diminished respiration in left lung, and numbness of left arm. Hæmoptysis, aphonia, and dysphagia. Aneurism projecting beneath

\* Mr. W. B. died in a paroxysm of dyspnœa while this sheet was going through the press. The body was examined by Dr. Nixon, in presence of Dr. Brady, Mr. Brady, and myself. A large true aneurism, of tubular figure, and quite empty, was found to engage the entire of the ascending portion of the arch of the aorta.



sternal extremity of left clavicle, characterized by a double impulse, and likewise two sounds, the latter of which was sharp and accentuated. The sounds were audible on both sides posteriorly. No evidence of cardiac disease. The annexed tracing of the radial pulse, which was abrupt, but regular, equal in the two arms, and 96 in the minute, was taken by Dr. Nixon. It indicates rigidity of the arteries without hypertrophy of the left ventricle.

CASE CL.—Through the courtesy of my colleague Dr. Nixon, I have been favoured with the opportunity of observing and repeatedly examining a remarkable case of thoracic aneurism, which was admitted into hospital under his care, in January, 1875. The patient, a man aged forty-one years, and intemperate, had been attacked, two years previously, with pain shooting out at the left scapula, and extending round the chest and into the abdomen. He had subsequently repeated paroxysmal seizures of this character. During one of these attacks, and seven days before admittance, the left side of the chest became prominent, and ninety-six hours later, a large tumor appeared in the left scapular region. When first examined in hospital, this tumor occupied the situation of the scapula, which had been displaced backwards and outwards. It was hemispherical in figure, hyperæmic on the surface, and extended from the level of the second to the eighth dorsal vertebra, being fourteen inches in the vertical and nine in the transverse diameter. It yielded a single systolic impulse and a double sound. Pulsation subsequently became visible in the left supra-mammary region, and here respiratory sounds were absent, though elsewhere normal. He had been suffering from bronchitis, with huskiness of voice and cough, which gradually improved as the tumor projected behind, and ultimately disappeared. No dysphagia, or laryngeal phenomena. Precordial dulness was extended. A double murmur existed at apex, with clicking perceptible to the hand, and accompanied by pain and epigastric tenderness. These phenomena, which were due to intercurrent pericarditis, gradually disappeared. On the day after admittance, a slight prominence appeared beneath the sternal end of the right clavicle, and here, percussion-dulness existed over an area three inches in dia-

meter. This prominence yielded a systolic impulse and a rough systolic murmur, the latter of which was transmitted into the transverse portion of the aorta. The second sound was accented in the situation of the subclavicular prominence, and, as the stethoscope was moved from this point towards the heart, the murmur gradually faded away.

On the 27th of February, respiration was tranquil, and the cardiac phenomena were normal. Half a grain of morphia was given every third hour to relieve pain, with complete success. This man remained in hospital for several months, during which he was strictly confined to bed. He was supported in the semi-recumbent posture during the day. His diet was generous, but the allowance of liquid was limited. The tumor became solid, and the aneurismal sounds inaudible, but the impulse remained. The man's general health was much improved. He became impatient of further restraint, and left hospital unexpectedly in the end of May.

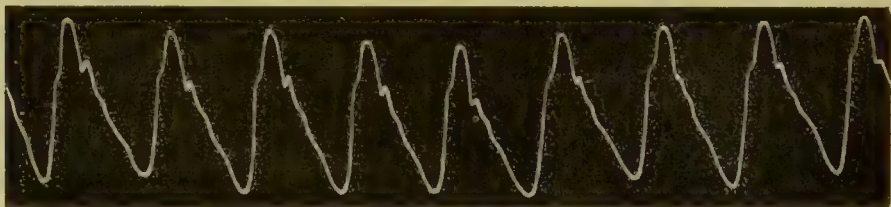


FIG. LXXVIII.

Tracing of posterior aneurism. Peter S. Pulse 84.  
February 20th, 1875.

The above tracing from the posterior aneurism was taken by Dr. Nixon, and kindly placed at my disposal.

*Aneurism of the Abdominal Aorta* occurs most frequently in middle age, and is usually traceable to a definite accident, such as a strain in lifting a heavy weight, or a blow upon the abdomen or back. It is more common in the male than in the female sex. The earliest age at which it occurred amongst the cases given in Table XVII. was eighteen years, and the latest, fifty years; and, of the 16 cases epitomized in that Table, only 1 was in the person of a female.

Of the 59 cases collected by Dr. Crisp, the ages ranged from twenty to sixty years (33 being under forty years), and of this

number only 8 were females.\* Since the publication of Crisp's work (1847), seven additional cases in the female have been adduced from British medical records by Dr. A. W. Foot, including one published by himself.† Most probably, however, the ratio, as between the sexes, has not been much if at all deranged within the period mentioned.

The abdominal aorta, in any part of its course, may be the seat of aneurism. The upper portion of the vessel, between the opening in the diaphragm and the origin of the superior mesenteric artery, is, however, its most frequent point of origin. Out of 177 cases of aneurism of the abdominal aorta analyzed by Dr. Sibson, the portion of the vessel at or near the cœliac axis was the seat of the disease in 131 instances.‡ The vicinity of the cœliac axis may be regarded as the point of election. Hence, the solar plexus is usually involved in, or stretched upon, the tumor, a circumstance to which is due the excruciating pain, radiating through the abdominal viscera, by which aneurism in this situation is distinguished. When situated lower in the abdomen, the aneurism is attended with less urgent symptoms, and is less obscure.

Abdominal aneurism is usually of the false variety, and, as contrasted with thoracic aneurism, is less frequently associated with extensive atheroma of the aorta, and with fatty or other structural disease of the heart. It is likewise unaccompanied by fever, and, with the exception of pain, by less formidable symptoms, and it rarely proves fatal by excentric pressure. The exceptions are, death by exhaustion from excessive pain produced by nerve-pressure or by inanition; from obstruction of the œsophagus, the pylorus, or the thoracic duct.

The symptoms of abdominal aneurism may be now discussed in detail; and first, those which characterize the disease originating in the aorta.

Pain is the most frequent, the most urgent, and usually, the first symptom. It is of two kinds, as pointed out by Dr. Law,§ both of which, however, are not necessarily associated in the same case. The usual pain, and that which constitutes the earliest

\* *Structure, Diseases, and Injuries of the Blood Vessels*, p. 161.

† *Dublin Quarterly Journal of Medical Science*, vol. li., February, 1871.

‡ *Medical Anatomy*, pp. 57, 58.

§ *Dublin Journal of Medical Science*, vol. xxi., 1842, and vol. xxv., 1844.



and the most prominent symptom of the disease, is characterized by sudden accessions of the most extreme severity, closely resembling the paroxysms of painter's colic. It radiates from the epigastrium, through the abdomen, into the pelvis, back, and lower portion of the chest; and then, after an indefinite period, usually not exceeding a few hours, quite as suddenly ceases, and leaves the patient exhausted, but free from actual suffering. There is, during the intervals, a total absence of febrile excitement or constitutional disturbance. Accessions of this character are usually determined by causes which produce excitement of the circulation, and increase of vascular pressure. Hence, exercise or emotion, a full meal, copious imbibition of liquids, or the use of stimulating food or drink, is the ordinary cause of an attack. One of the first recorded cases in which pain of this violent and paroxysmal character was associated with aneurism of the abdominal aorta, was observed by Bertin in 1805.\* But the most striking example of this kind on record is the well known case published by Dr. Beatty.†

The most urgent pain of this character, is associated with aneurism of the portion of the aorta embraced by the crura of the diaphragm, and has reference rather to the tension produced by the tumor than to the direction of its growth. In this situation, the great splanchnic nerves are exposed to compression, and in a degree proportionate to that in which the aneurism is restrained in its forward and lateral expansion. The constituent branches of the solar plexus, stretched between the semi-lunar ganglia and the celiac axis, are likewise exposed to tension from an aneurism of the aorta immediately below the diaphragm. Hence, paroxysms of pain radiating through the abdominal viscera, with spasm of the abdominal muscles, and temporary inflation of the bowels, so often witnessed. This pain may be reflected in the course of the ureter, to the testicle or groin, and be readily mistaken for that produced by the passage of a renal calculus. From this, however, it may be distinguished by the absence of hæmaturia.

The second kind of pain above referred to is dull and boring in character, localized, and continuous. It is associated with

\* *Opus citat.*, p. 115, observation 42.

† *Dublin Hospital Reports*, vol. vi., 1830.



tenderness at a corresponding point of the spinal column behind, and is all but pathognomonic of erosion of the vertebræ. Not unfrequently connected with this, is a "nipping" pain in the course of the intercostal nerves.\* The probability of spinal hyperæsthesia, especially in the female, and the vague and anomalous character of the pain, which must be accepted on the patient's representation, diminish in some degree the value of this symptom. If, however, the pain be aggravated by stooping, or by stamping; by jumping from a height, or by rotating the body whilst standing upon one leg, it may be regarded, if associated with the symptoms and signs of aneurism, as indicating erosion of the vertebræ. In many cases, relief from this pain is obtained only in the prone or stooping posture, and the patient instinctively lies upon his face or leans forward, with his elbows or his head supported in front. Abdominal aneurism located beneath the crura of the diaphragm is rarely unattended by erosion of the vertebral column. With this exception, Laennec was right in asserting that the vertebræ were less frequently eroded by an abdominal than by a thoracic aneurism, owing to the "free surroundings" of the former.†

Jaundice may result from pressure upon the biliary ducts, or venous congestion of the lower half of the body, from compression of the inferior cava. The former of these symptoms is, however, more frequently associated with aneurism of the superior mesenteric artery than of the aorta. Amongst the rare symptoms of abdominal aneurism, whether of the aorta or one of its branches, obstruction of the œsophagus below the diaphragm by the pressure of the tumor;‡ nausea and vomiting from pressure upon the pylorus; forward displacement of the liver, and upward displacement of the heart may be mentioned.

The pulse rarely exhibits any special characters in connexion with abdominal aneurism. It may, however, be intermittent, di-crotic, hyperdicrotic, or, as pointed out by Dr. W. Wade, even tricrotic; the secondary wave exceeding the first in altitude.§

\* *Vide* a case by Dr. Law, *Dublin Journal*, vol. xxii., p. 388.

† *Opus citat.*, tom. ii.

‡ *Vide* a case by Sir D. Corrigan, *Dublin Journal of Medical Science*, vol. ii. January, 1833.

§ *British Medical Journal*, January 4th, 1868.

I believe that tricotism can exist only in connexion with an aneurism of large capacity, the secondary and major wave being due to reflux from the sac, the walls of which, by their reaction, force a large volume of blood back into the aorta after arterial systole.

*The physical signs* consist in impulse, dulness, sound, thrill, and murmur. The impulse of abdominal aneurism is single, systolic, and heaving. It may be mistaken for strong pulsation of the aorta from (a) spanæmia, (b) previous hæmorrhage, (c) hysteria, (d) acute dyspepsia, (e) menstrual excitement, (f) the quickening of pregnancy, and (g) intestinal or peritoneal inflammation. It is still more likely to be confounded with an impulse communicated to a tumor overlying the aorta.

For the purpose of arriving at a positive diagnosis, the patient should be placed in the recumbent posture, the anterior abdominal muscles being relaxed to the uttermost by flexing the hip and knee joints. The seat of pulsation should be then carefully explored, by means of the fingers and thumb placed on opposite sides of the aorta, the dimensions of which, and the extent of its pulsation, may be determined by this expedient. If the aorta be found of uniform diameter throughout, and to pulsate equally over its entire length, aneurism will be virtually excluded. It may be necessary to administer chloroform for the purpose of producing complete relaxation of the muscles. In cases of simple pulsation of the aorta, vascular excitement will be found to extend to the iliac and femoral arteries, whereas, the pulsation of aneurism is strictly local; and, finally, under chloroform or other anæsthetic, the former will subside, the latter will not. Sympathetic excitement of the aorta, from inflammatory irritation of the bowels or peritoneum, will be characterized by dysenteric symptoms, or acute sensibility to touch, and by rise of temperature as determined by the thermometer.

The distinction between communicated pulsation, and that of an aneurism, which is often attended with great difficulty, will be more conveniently considered after the physical signs of aneurism have been fully discussed.

An aneurism of the abdominal aorta most frequently springs from the anterior wall of the vessel. When situate, as it usually

is, in the epigastrium, it projects to the left of the middle line in most instances, and, in the progress of growth, has a tendency to *descend*. When discoverable by palpation, it presents in most cases the character of a smooth, elastic, and pulsating tumor, communicating to the hand alternate movements of lifting, combined with uniform expansion and progressive tension, and of subsidence with relaxation. The pulsation is almost invariably single, and slightly posterior in time to that of the heart and the carotid arteries, but synchronous with the radial pulse;\* it is occasionally accompanied by thrill. Pressure upon the aorta below the tumor will increase the force of impulse, diminish or abolish the thrill, and arrest the collapse. In a case of thoracico-abdominal aneurism mentioned by Stokes, in which the heart had been displaced upwards and to the right by the pressure of the tumor, the latter yielded a double pulsation and sound, both probably communicated from the heart.† In a few instances the tumor has been hard, rugged, and non-expansile, the sac having been uneven on the surface, and lined by solid laminated fibrin.‡ It may yield no pulsation whatever, even though containing fluid blood, a circumstance which Pirogoff would attribute to the impaction of a solid clot in the mouth of an aneurism with a narrow aperture, or to the compression of the vessel by the aneurism itself, on the proximal side of the sac.§

The evidence elicited by percussion is less conclusive in regard to abdominal than thoracic aneurism. This is due to the deep situation of the tumor, with the intervention of the stomach or intestines in front, and to the presence of the mass of lumbar muscles behind. If, however, the anterior muscles be relaxed, and the stomach and intestines free from flatus, percussion of the tumor will elicit absolute dulness.

Single or double sound, as distinguished from murmur, is rarely heard in front in connexion with abdominal aneurism. It did not exist in any of the cases comprised in Table XVII.

\* *Vide* Case 137.

† *Opus citat.*, p. 618.

‡ *Vide* a case by Dr. James Little, *Dublin Journal of Medical Science*, October, 1872; also a case by Dr. James S. Hughes, *Medical Press and Circular*, September 10th, 1873.

§ Quoted by Holmes, *St. George's Hospital Reports*, 1875.

Though not audible in front, sound, usually double, is occasionally heard over a limited portion of the dorsal or lumbar spine. When so limited, and associated with other evidence of aneurism, it is of the utmost diagnostic value.

Thrill is a rare phenomenon in abdominal aneurism. When present, it coincides with the impulse, and is due to vibration of the walls of the sac. Murmur is, next to impulse, the sign most frequently connected with aneurism of the abdominal aorta. It is single, postsystolic, and prolonged, and in quality soft and blowing. It is not propagated in the course of the aorta. If the aneurism arise from the anterior wall of the vessel, the murmur may be audible only in front; and if, from its posterior wall, it may be heard only at a corresponding point in the back. When the aneurism is large, presenting a perceptible tumor in front, and pressing at the same time upon the vertebral column, the murmur will most probably be heard both in front and behind. It occasionally happens that, whilst a murmur is audible in the epigastric or umbilical region, a single or double sound, without murmur, is heard over the corresponding portion of the vertebral column. A few cases are on record in which an aneurism of the abdominal aorta has yielded a double murmur. One such has been published by Dr. Alexander Fleming; in this case a systolic and diastolic murmur were audible at the epigastrium, and a systolic murmur with two sounds in the left loin.\* Dr. Willoughby Wade has also published a case in which a double murmur was associated with a single impulse.† The quality of the murmur is sometimes sharp or musical, or it may be shrill in one portion of the tumor, and blowing in another. It is occasionally of an intensely buzzing character, the sac being lax and devoid of coagulum, and therefore capable of strong vibration.‡

The murmur of abdominal aneurism is singularly influenced by posture. As first noticed by Sir Dominic Corrigan, it is suspended when the patient assumes the erect posture; an effect which he attributes to tension of the sac by the weight of the

\* *An Inquiry into the Physiological and Medicinal Properties of the Aconitum Napellus*, 1845.

† *British Medical Journal*, January 4th, 1868.

‡ *Vide* a case by Dr. Roberts Archer, *Irish Hospital Gazette*, January 1st, 1875.



column of blood above it. He adds, however, that murmur will not exist, even in the recumbent posture, when the sac is lined by thick layers of fibrin, and therefore incapable of reacting upon its contents.\* Cessation in the erect posture is an all but invariable characteristic of this murmur, and the theory propounded by Sir D. Corrigan in explanation of it, is most probably correct. In a few recorded cases, however, murmur has been heard both in the recumbent and the erect posture; and in a case of large abdominal aneurism recently published by Staff-Surgeon Adsetts, a loud murmur was heard in the erect posture, which ceased when the patient lay down.† These anomalies have reference, most probably, to special conditions of the sac and its contents, upon which, in the present state of knowledge, it would serve no useful purpose to speculate.

Abdominal aneurism may be *latent* in regard to physical signs, whilst associated with severe paroxysmal pains, eminently suggestive of aneurism.‡ In such cases a positive diagnosis cannot be made, and an aneurism of small or medium size, arising from the posterior wall of the aorta, will be found to have eroded the vertebral column.

True aneurism of the abdominal aorta, which is of extreme rarity, may be unattended by either signs or special symptoms. A case of true aneurism of the aorta, above the bifurcation, of the size and shape of a pear, in which no tumor could be detected and no pain or tenderness existed, has been published by Dr. Arthur W. Foot. Rupture of the sac was announced by sudden aching pain in the left iliac region, with cramps and pain in the lower extremity, followed by the appearance of a large and rugged tumor in the left iliac, lumbar, and hypochondriac regions. Copious hæmorrhage had taken place behind the peritoneum.§

The *diagnosis* of aneurism of the abdominal aorta may be now briefly considered, with reference to the preceding symptoms and signs viewed in the concrete.

\* *Lancet*, April 11th, 1829, and *Dublin Journal of Medical Science*, vol. ii., January 1st, 1833.

† *Irish Hospital Gazette*, April 15th, 1874.

‡ *Vide* a case by Dr. Holmes, *St. George's Hospital Reports*, 1875, p. 191.

§ *Dublin Quarterly Journal of Medical Science*, vol. li.

It is most frequently presented as a smooth pulsating tumor in the epigastrium and to the left of the mesial line, exhibiting a downward growth, and not movable by manual pressure, or by forced inspiration. The symptoms most frequently exhibited are, pain of the twofold character previously described, and local tenderness; whilst the usual physical signs are, a single impulse accompanied by a soft and prolonged bellows-murmur, both slightly posterior in time to the impulse and the first sound of the heart. In exceptional instances a second murmur, and still more rarely, a second impulse of diastolic or postdiastolic rhythm, has been detected. In the back, a single or double sound, without impulse or murmur, are the signs usually exhibited. But a single impulse and a single murmur of postsystolic rhythm, the latter usually associated with a double sound, and not necessarily identical in tone or quality with the corresponding murmur at the epigastrium, are in many cases discoverable over a limited portion of the vertebral column. In a case mentioned by Dr. Stokes, the patient experienced relief from pain when he lay on either side, whilst the tumor and the impulse became imperceptible; when he turned upon his back, he was immediately again attacked with pain, the tumor and the impulse at the same time gradually returning.\*

The murmurs, both epigastric and dorsal, are all but invariably suspended in the erect posture, whilst they are usually in no degree influenced by the prone position,† as pointed out by Dr. Stokes, and more recently urged by Dr. William Moore.‡ As regards general symptoms, aneurism within the abdomen is characterized by absence of constitutional irritation. Febrile reaction is of rare occurrence; it is exhibited only towards the close of protracted cases, and is then of a low or irritative character, associated with wasting from constant pain, and loss of appetite and sleep.

The diseases and conditions for which aneurism in the abdo-

\* *Proceedings of Pathological Society of Dublin*, December 14th, 1844.

† In a case of abdominal aneurism published by Dr. James S. Hughes, to which further reference will be made, both the impulse and the murmur ceased in the prone position; see *Medical Press and Circular*, September 10th, 1873.

‡ *Dublin Quarterly Journal of Medical Science*, vol. xlix., 1870, p. 479.

men is most likely to be mistaken, are: (a) cancerous or other tumor, receiving a pulsation from the aorta; (b) an engorged liver, receiving a reflux pulsation from the right ventricle through the inferior cava (Burns\*); or an enlarged left hepatic lobe, exhibiting the same phenomenon from contact with the aorta (Stokes); (c) an enlarged pancreas similarly circumstanced (Stokes, Burns); (d) psoas abscess; and (e) cystic tumor (Holmes, Pirogoff,† Bakewell‡).

A cancerous tumor in contact with the aorta, and exhibiting a communicated pulsation, has been frequently mistaken for aneurism. Dr. O'Ferrall has reported a case in which a malignant growth in the epigastrium, situate behind the aorta, gave rise to murmur and strong pulsation, similar to those of an aneurism.§

I have met with a somewhat similar case. A man, aged fifty-six years, suffered from severe and continuous pain in the epigastrium, with occasional hæmatemesis; a pulsating tumor, with a loud bellows-murmur, existed in the seat of pain. The patient was emaciated, and died from repeated hæmorrhage. The tumor was found to consist of scirrhus of the pancreas. The duodenum, where in contact with the pancreas, had been ulcerated through, and an opening had been formed in the pancreatico-duodenalis artery, from which the hæmorrhage had proceeded.

Aneurism, on the other hand, is liable to be mistaken for a cancerous tumor. Dr. James Little has published the case of a woman, aged forty-nine, in which a large aneurism of the aorta above the origin of the renal arteries, presented the character of a hard non-expansile tumor, which was lifted *en masse* with each pulsation of the vessel. The difficulty of the case was still further enhanced by the existence of open cancer of the cheek, of which the patient ultimately died. Most of the symptoms and signs of aneurism were, however, well pronounced, and the diagnosis of aneurism was made.||

In the case published by Dr. James S. Hughes, already referred to, there were, in addition to the cessation of impulse and

\* *Observations on the Diseases of the Heart*, 1809, p. 270.

† Quoted by Holmes, *loc citat.*

‡ *Royal Medical and Chirurgical Society*, October 27th, 1874.

§ *Pathological Society of Dublin*, January 19th, 1850.

|| *Dublin Journal of Medical Science*, vol. liv., 1872.

murmur in the prone position, the further misleading features of a hard and rugged tumor, and a family history of cancer. Nevertheless, the diagnosis of aneurism was made.

The special characteristics of cancer may be now briefly noticed.

There is, not unfrequently, a family history of cancer, and evidence of the disease on the exterior of the body. The symptoms are steady in progress, and not much, if at all, modified by treatment. There is a tendency to upward, rather than downward growth of the tumor; the pain is seldom severe, and never paroxysmal; the patient is emaciated at an early period of the disease, and is singularly apathetic throughout. Finally, varicose enlargement of the superficial veins, and ascites, are ordinary symptoms, friction-sound may be occasionally detected, and the associated murmur is not influenced by change of posture. With the exceptions previously noticed, aneurism contrasts with cancer in respect of all the foregoing characteristics.

An aneurism which has become diffused by rupture of the sac, may closely resemble a psoas abscess; the extravasated blood, having infiltrated the retro-peritoneal tissue, may present a fluctuating tumor in the lumbar region, or the groin, where psoas abscess usually points. The existence of localized pain and tenderness of the spine will tend still further to mislead. Diffused aneurism, however, usually retains a faint impulse and murmur, whilst femoral pulsation on the affected side is diminished or suspended from pressure of the effused blood upon the artery at some point below the aneurism, or from blocking of the common iliac by a displaced fragment of clot at the moment of rupture of the sac, as occurred in the case published by Dr. Foot, and previously referred to. Interrupted circulation in the lower extremity may likewise serve to distinguish the pain and collapse due to rupture of an aneurism, from the same symptoms when caused by perforation of the stomach or intestine, as shown by Dr. Stokes.

Mr. Holmes has recorded an example of medullary cancer of the kidney, which yielded visible pulsation in the loins, with a loud *bruit*. A correct diagnosis was, however, made from the twofold circumstance, that the pulsation was not in proportion



to the size of the tumor, and that the murmur did not possess the "rushing" character of that of an aneurism.\*

*The celiac axis* and its primary branches, and the *superior mesenteric artery*, are frequently the seat of aneurism. In regard to differential diagnosis, aneurism of these vessels is related to that of the abdominal aorta, as innominate aneurism is to that of the arch. It therefore demands a brief notice here. Examples of superior mesenteric aneurism have been published by Hughes Bennett,† Gairdner,‡ Elliotson, Douglas,§ O'Ferrall,|| and Dr. Arthur Wilson (two cases).¶

In some of these cases, a physical examination had not been made, and the disease was identified only after death; but in others a superficial pulsating tumor, yielding a bellows-murmur, and characterized by great mobility, and by change of position as the patient turned from one side to the other, was recognized. Severe pain in the epigastric and umbilical regions existed in these cases, and in most of them, likewise, jaundice, more or less pronounced, from pressure upon the hepatic or the common biliary duct, was exhibited. In the case published by Dr. Gairdner, together with jaundice, two of the symptoms of gastric ulcer existed; viz., vomiting after meals, and repeated hæmatemesis. Nearly two years later, death occurred by rupture of the sac into the peritoneum. It was then found that long previously the aneurism had opened into the duodenum, whence the hæmatemesis had proceeded.

In a case of aneurism of the hepatic artery recorded by Dr. Stokes, hæmatemesis and persistent jaundice were likewise prominent symptoms; the former, as in cirrhosis of the liver, obviously by exhalation of blood into the stomach, as no direct communication between the sac and the alimentary canal existed; and the latter, from pressure upon the common bile-duct. Death was caused by rupture of the aneurism into the peritoneum. The liver was found to be reduced in volume, whilst

\* *Loco citat.*

† *The Principles and Practice of Medicine*, second edition, p. 577.

‡ *Clinical Medicine*, p. 495.

§ Quoted by Gairdner, *opus citat.*

|| *Proceedings of Pathological Society of Dublin*, March 16th, 1850.

¶ *Medico-Chirurgical Transactions*, vol. xxiv., p. 221.

the gall-bladder and the hepatic ducts were distended with bile.\* In one of Dr. Wilson's cases, nearly all the structures and fluids of the body were deeply bile-stained, and the biliary ducts throughout the liver were enlarged to the size of the cystic duct; in the other, hæmatemesis and melæna were the most prominent symptoms.

From the evidence furnished by the preceding cases, it would appear that the distinctive characteristics of aneurism of the primary branches of the abdominal aorta, are, mobility of the tumor, and the symptoms of pressure upon (a) the bile-ducts, (b) the portal vein, and (c) the stomach, pylorus, or duodenum.

The duration of life with abdominal aneurism is quite indefinite, and no less variable than in the thoracic form of the disease. In the 16 cases tabulated (Table XVII., p. 1216), it ranged from fifteen days to eleven years.

Death is most frequently caused by rupture of the sac, and effusion of blood behind the peritoneum, into the peritoneal cavity, or into the left pleura. In 7 out of the 16 cases just referred to, a secondary diffuse false aneurism was formed by rupture of the sac behind the peritoneum, the patients having survived this accident for periods varying from a few hours to several weeks. In one of these cases (No. 8) there was a secondary irruption of blood into the cavity of the left pleura.

Extravasation into the left lung or pleura occurred in 3 instances, including that just mentioned, and into the cavity of the peritoneum in 1. Death resulted from syncope without rupture of the sac in 2 cases, from exhaustion in 1, and from Bright's disease in 1.

Of the 47 cases of abdominal aneurism collected by Crisp,† in which the mode of death is stated, 11 opened behind the peritoneum, 10 into the peritoneum, 5 into the left pleura, 3 into the inferior cava, 1 into the lung, 1 into the colon, 1 into the the pelvis of the kidney, 1 into the posterior mediastinum, 1 "into the chest." In 13 instances death occurred without rupture of the sac.

\* *Dublin Journal of Medical Science*, first series, vol. v.

† *Opus citat.*, p. 163.

*The treatment* of abdominal aneurism should be conducted on the same principles as that of the thoracic form of the disease. The medicinal agents already mentioned, as useful in the treatment of thoracic aneurism, whether with a view to alleviation or cure, are equally available here.

For relief from pain, large doses of opium or morphia are demanded, and may be given in gradually increased quantity, without fear of narcotism. In one of Dr. Little's cases, already referred to, *twelve and a-half* grains of the acetate of morphia were administered twice daily by hypodermic injection, that is, *twenty-five* grains a-day for some time, without inducing narcotism.

In a case of thoracic aneurism reported by Assistant Surgeon Boileau, which opened into the right bronchus, the patient, an old soldier, took half an ounce of laudanum daily for fifteen days, during which he survived the first hæmorrhage.\*

An example of cure of an abdominal aneurism in twenty-five days, under treatment with the acetate of lead (three grain doses, with a quarter of a grain of opium, thrice daily), aided by rest in the recumbent posture, and a restricted diet, has been reported by Dr. Daly.† I incline to think that rest, rather than the medicine used, should be credited with the favourable result of treatment in this case. The success which, in the hands of Mr. Tufnell, has attended the treatment of aneurism by means of rest and regulated diet, without specific medication, would seem to warrant this conclusion. This plan of treatment, which has been already discussed (p. 1141) is no less applicable to abdominal than to thoracic aneurism.

The treatment by compression has, in several instances, been attended with the most gratifying results. The first recorded example of success by this means, was published by Dr. William Murray, of Newcastle-on-Tyne, to whom the great merit of having introduced this mode of treatment, as applied to abdominal aneurism, belongs.‡ In this case, an aneurism of the abdominal aorta involving the origin of the inferior mesenteric

\* *Army Medical Report*, 1867, p. 325.

† *Biennial Retrospect of Medicine and Surgery*, 1865-6, p. 305.

‡ *Medico-Chirurgical Transactions*, vol. xlvii., 1864; and *The Rapid Cure of Aneurism by Pressure*, 1871.

artery, was cured by proximal compression of the aorta, the patient being under the influence of chloroform, within the short period of one hour. Two *séances* were given; the first, which lasted two hours, was unsuccessful; but the second, three days later, and continued for five hours, during the last hour of which circulation through the sac was completely arrested, resulted in perfect solidification and cure. The patient died six years afterwards, and both the diagnosis and cure were verified by dissection. The instrument used in this case was the ordinary horse-shoe tourniquet. A second case, in which cure was effected by compression in three quarters of an hour, has been reported by the same physician.\* Dr. Murray has likewise mentioned a case treated by Dr. Heath, in which cure by compression was effected in *twenty minutes*.†

Doctor Moxon and Mr. Durham have published a successful case. Compression by means of Lister's "abdominal tourniquet" was maintained under the influence of chloroform for ten hours and a-half; gradual solidification of the aneurism followed, and in little more than a month after the application of pressure, the pulsation of the sac had quite ceased. Four months later the patient was in good health, and the remnant of the aneurism, reduced in size, solid, and free from pulsation, was felt.‡

With a view to effecting immediate coagulation in the sac, as the most favourable condition for solidification of its contents, Dr. O'Ferrall has recommended that distal compression should precede and accompany proximal compression of the artery.§ In a communication of great interest and value, Dr. Mapother has reported the successful application of this principle in his own practice. In a case of large ilio-femoral aneurism in which proximal compression alone had failed, pressure applied simultaneously both above and below the aneurism under the influence of chloroform, effected a perfect cure in four and a-half hours.|| The instruments used by Dr. Mapother were those

\* *British Medical Journal*, October 5th, 1867.

† *Ibid.*

‡ *Medico-Chirurgical Transactions*, vol. lv., 1872.

§ *Dublin Medical Press*, March 15th, 1865.

|| *Ibid.*, March 29th, 1865.



known as Skey's and Signorini's tourniquets. In a case of right ilio-femoral aneurism published by Mr. Holden, pressure by means of Lister's tourniquet upon the aorta at its bifurcation, but, as in Dr. Mapother's case, bearing mainly upon the right common iliac, effected solidification of the contents of the sac in four hours.\*

For treatment by proximal compression, it is necessary that sufficient space shall exist for the application of effective pressure to the aorta between the diaphragm and the aneurism. Where such is not the case, distal pressure, as practised by Mr. Bryant,† may be tried. The pressure must be such as completely to shut off circulation through the sac, with a view to rapid coagulation of its contents. Before it is commenced, the bowels should be well moved, and freed from flatus, and during its continuance, chloroform or other anæsthetic must be administered. The procedure, though apparently harmless, involves some risk of injury to the bowels. In Mr. Bryant's case, death occurred eleven hours after the experiment, from entero-peritonitis produced by the pressure of the tourniquet.

In none of the cases of abdominal aneurism, four in number, which have come under my notice, has death occurred whilst they were under my observation. In the absence of verification by autopsy, a history of these cases, which was that of aneurism of the abdominal aorta in its typical form, would contribute nothing to the advancement of science, whilst it would add to the bulk of a work already too large.

The case published by Dr. James Stannus Hughes, and previously referred to, claims special notice here, both on account of its intrinsic value to science, and of its melancholy associations. The following abstract of the case, the details of which, as given by Dr. Hughes,‡ are well worthy of perusal, will suffice to exhibit its principal features. An eminent surgeon, resident in Dublin, and aged fifty-four, consulted Dr. Hughes in May, 1867, with reference to severe pains in the loins and sacrum, and ex-

\* Mr. Eck, in *St. Bartholomew's Hospital Reports*, vii., and *Biennial Retrospect of Sydenham Society*, 1865-6.

† *Medico-Chirurgical Transactions*, vol. lv.

‡ *Medical Press and Circular*, September 10th, 1873.

tending to the groins and thighs, from which he had been only suffering from the previous evening. Ten years prior to the above date, this gentleman, whose habits had been strictly regular, was supposed to be the subject of a gastric affection, which was accompanied with pulsation of the abdominal aorta. For years he had presented the aspect of a person suffering from malignant disease; was wasted, discoloured, and haggard. His elder brother had died of cancer. For several years also he had had, in the abdomen, a pulsating tumor which he desired to conceal, and in regard to which he refused to admit a thought of aneurism. This tumor was of the size of a man's fist, hard and irregular on the surface, and, in the recumbent posture, pulsated with the up and down movement of a solid tumor overlying a large artery, rather than with the general and uniform expansion of an aneurism. It yielded a systolic bellows-murmur, but in the prone position both impulse and murmur ceased.

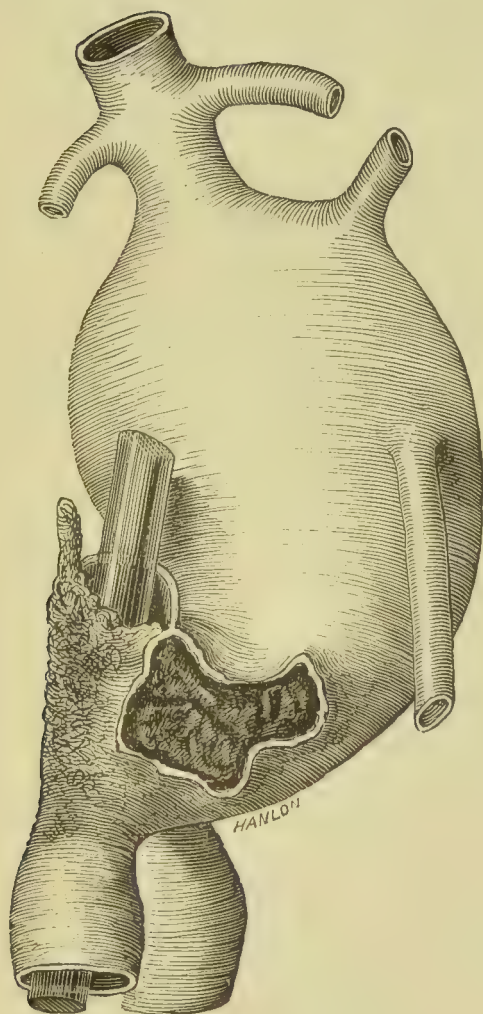
Three days subsequently to Dr. Hughes's first visit, and a few minutes after the patient had freely manipulated the tumor with the view of convincing his medical attendants "that it was not aneurism," it gave way, and death from hæmorrhage into the peritoneum followed within three or four minutes.

A large aneurism was found arising from the aorta immediately above its bifurcation. It had grown from the anterior wall of the vessel, and was nearly full of laminated and nodular fibrin, which had given it the rugged feel previously mentioned. Its anterior wall was thin, and had given way in the two places. Two of the lumbar vertebræ were in an early stage of caries. The annexed engraving (Fig. LXXIX.), for the use of which I am indebted to the kindness of Dr. Hughes, exhibits the aneurism as presented after dissection.

Dr. Hughes directs special attention to the following, amongst the many features of interest presented by this case; viz., the long duration of the disease; the absence of pain till three days before death; the unusual character of the pulsation, and the absence of pulsation and murmur in the prone position.

Table XVII. has been constructed from the records of the Pathological Society of Dublin.

FIG. LXXIX.



Aneurism of abdominal aorta. (From Dr. James S. Hughes.)

TABLE XVII.]

## ANEURISM OF THE ABDOMINAL AORTA.

No.	Author.	Sex, Age, and Occu- pation.	Habits and Cause of Aneurism.	Duration in Months, and Seat of Aneurism (when stated).	General Nutri- tion.	Aneur. Mur- mur.	Aneur. Sounds.	Aneur. Impulse.	Mode of Death.
1	Dr. Law	Gentle- man, 38	Temperate; occasional falls in hunt- ing-field	12 : Abdom. aorta, posterior wall, at coeliac axis	Good	Systolic	None	Systolic	Rupture to ret periton tissue
2	Dr. Stokes	Gentle- man	..	18 : Abdom. aorta between crura of diaphragm	Good, until shortly before death	Systolic	None	Systolic	Rupture to lun
3	Dr. O'Ferrall	Male, 18	Strain in wrestling	Some mts. : Abdom. aorta betw. crura of diaphragm	..	Systolic	None	Systolic	..
4	Dr. Greene	Female	Undefined in- jury	132 : Abdom. aorta near superior me- senteric artery	..	Systolic	None	None	Rupture to ret periton tissue
5	Dr. Hutton	Male, 50	..	Some mts. : Abdom. aorta low down	..	Systolic	None	Systolic	Syncope
6	Dr. Law	Shoema- ker, 33	..	14 : Abdom. aorta between crura of diaphragm	..	Systolic	None	Systolic	Rupture to ret periton tissue
7	Dr. Law	Male	..	Abdom. aorta at origin of coeliac axis	..	Systolic	None	Systolic	Rupture to ret periton tissue
8	Dr. Law	Labourer, 35	..	36 : Abdom. aorta at coeliac axis	..	Systolic	None	Systolic	Rupt. in left ple & behi perito.
9	Dr. Stokes	Printer, 40	..	12 : Abdom. aorta between crura of diaphragm	..	Systolic	None	Systolic	Rupt. in periton cavity
10	Dr. O'Ferrall	Stonecut- ter, 34	..	18 : Abdom. aorta just below dia- phragm	..	Systolic	None	Systolic	Rupt. in l. lung pleura
11	Dr. E. Hamilton	Car-dri- ver, 38	Intemperate	Abdom. aorta in middle portion	..	Systolic	None	Systolic	Rupture to ret periton tissue
12	Dr. Gordon	Labourer in vitriol works, 50	..	15 days : Abdom. aorta and left c. iliac	..	..	..	..	..
13	Dr. McDowel	Dischgd. soldier, 29	..	14 : Abdom. aorta at bifurcation	..	Systolic	None	Systolic	Rupture to ret periton tissue
14	Mr. W. Stokes	Cattle drover, 42	Temperate; hardship	48 : Abdom. aorta below coeliac axis	..	Systolic	None	Systolic	Asthenia
15	Dr. W. Moore	Coal-hea- ver, 31	Fall from height	36 : Abdom. aorta below coeliac axis	..	Systolic	None	None	Syncope
16	Mr. Tufnell	Ship- wright, 31	..	Some time : Abdom. aorta below coeliac axis	Good	Systolic	None	Systolic	Bright's disease



## ANEURISM OF THE ABDOMINAL AORTA.

[TABLE XVII.]

<i>Direction of Growth.</i>	<i>Disease of Valves.</i>	<i>State of Ventricles.</i>	<i>State of Aorta.</i>	<i>Observations. General State of Heart.</i>
Backward and laterally	None	R. Normal L. Dilated and hypertrophied	Atheromat. in seat of disease	Slightly hypertrophied.
Upward	None	..	..	..
Forward and upward	None	..	..	Not hypertrophied. Caused palpitation by tilting forward heart.
Upward and to left	None	..	..	..
Upward, downward and to right	None	..	..	Pain in left testis and thigh. No lumbar or abdominal pain. Jaundice by pressure on hepatic duct. Disease at first suspected to be renal.
Forward	..	..	..	..
Backward	..	..	..	Disease at first taken for sciatica.
Backward and laterally	..	..	..	Constant aching or boring, and occasional darting, pain.
Forward	..	..	..	Sudden cessation of impulse in epigastrium. Pain ceased and tumor disappeared when he lay on side, but both returned when he lay on back.
Upward and to left	..	..	..	Murmur ceased in sitting posture. Had most ease while lying on back, although last dorsal and first lumbar vertebræ were found eroded.
Backward and downward	..	..	..	Fixed aching pain, varied by paroxysms shooting down thighs, to knees.
Course of vessels	..	..	Atheromatous	Dissecting aneurism engaging entire abdominal aorta, and left com. iliac, into which stream had re-entered.
Forward and upward	None	Normal	Atheromatous	Not hypertrophied. Lived ten days after rupture of sac and formation of a secondary false aneurism.
Backward	Aortic	R. Normal L. Dilat & hypert.	Atheromatous	Hypertrophy and dilatation.
Forward and to right	..	..	..	General hypertrophy. Aneurism had obliterated right renal artery. Right kidney was reduced to one ounce.
Forward	None	Normal	Atheromatous in seat of aneurism	Not hypertrophied. Aneurism cured by position, rest, and a restricted but nutritious diet, in thirty-seven days.

*Dissecting* aneurism, consists in a breach of continuity of the internal and middle coats of an artery, and a separation of these from the external tunic, to a variable extent over the length and circumference of the vessel, or a splitting of the middle coat, by the force of the blood-current. It is of two kinds: that with a single aperture, through which the blood enters the sac, and returns to the artery; and that which exhibits both an aperture of entrance and an aperture of exit.

The designation "dissecting aneurism" was first used by Laennec; but examples of this form of aneurism had been previously published by Morgagni,\* Nicholl,† Burns,‡ and Shekelton.§

Doctor Peacock has given an analysis of 19 cases of dissecting aneurism, including 5 not previously published.|| The ages of the patients, ascertained in 10 cases, ranged from twenty-four to eighty-four years; and of 15 in which the sex was stated, 10 were males, and 5 females. Dr. Crisp, in 1847, published a list of 21 cases, of which 14 were females, and 7 males; the ages of the patients varied from twenty-four to ninety years.¶

The ascending portion of the aorta, in the vicinity of the valves, is the usual seat of primary lesion. Such was its position in 10 out of the 19 cases analyzed by Dr. Peacock. In the majority of cases in which the disease is so situate, the coats of the aorta are tunnelled from its root to the arteria innominata, and death occurs by rupture into the pericardium. 15 of the 21 cases mentioned by Crisp terminated in this way; and of these, 7 died suddenly, 7 lived, one to twelve days after extravasation into the pericardium, one a month, and one "several months."

The case published by Laennec\*\* belonged to the first mentioned category, as did likewise most of those since recorded.

The second form of dissecting aneurism, which, as Dr. Peacock truly observes, represents an "imperfect natural cure" of the

\* *De Sedibus et Causis Morborum*, Epist. xxvi., § 15, 17, and 21.

† *Philosophical Transactions*, 1762.

‡ *Observations on Diseases of the Heart*, 1809.

§ *Dublin Hospital Reports*, vol. iii., 1822.

|| *Edinburgh Medical and Surgical Journal*, vol. lx., 1843.

¶ *Opus citat.*, p. 167.

\*\* *Opus citat.*, tom. ii., p. 696.

disease, is very rare. Mr. Shekelton, in 1822, published the two first recorded examples of it.\* They were obtained from subjects brought in for dissection, and nothing could be learned in regard to their history. In one, the aneurism occupied the last three inches of the abdominal aorta, and was formed by transverse rupture of the two internal coats on the anterior wall of the vessel. The abnormal channel lay in front of the aorta with which it communicated above by a semilunar opening, whilst below it was connected by two openings with the common iliacs; it was lined throughout by a dense layer of fibrin. In the second example, the walls of the left common iliac artery were channelled; the circulation to the lower extremity having been carried on through the abnormal, whilst that of the pelvis took the ordinary course through the internal iliac passage.

A somewhat similar example, but connected with a history of previous illness, was published by Dr. Henderson. The superior opening was situate near the left subclavian artery, and the inferior in the left common iliac.† Dr. Hyde Salter has recorded an example of dissecting aneurism of the ascending aorta with three openings into the vessel, in a girl aged twenty-four.‡ In this case, it would seem that the blood traversed the abnormal channel twice during each cardiac cycle; forwards with the systole, and backwards with the diastole of the left ventricle. I have met with one example only of dissecting aneurism.§

Aneurism of this form presents no special symptoms or signs by which it may be distinguished from ordinary saccular aneurism. Indeed, owing to the circumstance that it is usually cylindrical in figure, and of small diameter, the symptoms of excentric pressure are generally wanting, and the disease, in regard to symptoms, is not unfrequently latent. When, in the progress of a dissecting aneurism, symptoms of a special character are exhibited, they are strictly accidental, and therefore of no positive value for the purpose of diagnosis. Such were hemiplegia and violent pulsation of the carotid artery on the opposite side, in

\* *Dublin Hospital Reports*, vol. iii., p. 231.

† *London and Edinburgh Medical Journal*, July, 1843.

‡ *British Medical Journal*, December 31st, 1870.

§ *Vide Case 139*, p. 1177.

the case published by Dr. Todd.\* In that case, a dissecting aneurism of the ascending aorta and the arteria innominata, formed by splitting of the middle coat of the vessels into two layers, gave rise to atrophic softening of the right hemisphere of the cerebrum by pressure upon the common carotid artery; it likewise produced, incidentally, the phenomenon of violent arterial throbbing at the seat of vascular obstruction in the neck. In the case detailed by Dr. Hyde Salter, a diastolic murmur was developed at the aortic orifice by the reflux current through the aneurism descending, as he concluded, upon the ventricular surface of one of the sigmoid valves, and so preventing its closure.

*Stenosis of the aorta* may be either congenital or acquired. In the great majority of cases it is congenital, and situate at or near the junction of the ductus arteriosus. It was so situate in the 17 cases collected by Crisp,† and in 7 out of the 15 by Luton.‡ In the remaining cases of this last mentioned group it was located as follows: at the origin of aorta, 1; in transverse portion of arch (a double constriction), 1; in the abdominal aorta, 3; general coarctation, 3. In 3 of the cases analysed by Crisp, and in 2 of those by Luton, there was complete obliteration of the aorta. In the acquired form, it may result from local arteritis, with deposition and organization of fibrin at the seat of inflammation; or from the impaction and organization of a migratory clot.

As to form, the constriction may be linear, cylindroidal, or valvular.

According to Rokitansky, congenital narrowing near the ductus arteriosus results from perverted development of the branchial arches in this situation.§ Dumontpallier attributed it to extension, into the aorta, of the obliterative process by which the ductus arteriosus is closed;|| whilst Cheevers supposed it might be due to constriction of the aorta from tension of the left recurrent nerve, produced by the gradual ascent of the larynx into the neck with the first acts of inspiration.¶

\* *Medico-Chirurgical Transactions*, vol. xxvii., p. 301.

† *Opus citat.*, 1847, p. 31.

‡ *Nouveau Dictionnaire de Médecine et de Chirurgie*, vol. ii., 1865, p. 744.

§ *Pathological Anatomy*.

|| *Gazette Méd. de Paris*, 1857.

¶ *London Medical Gazette*, 1845, p. 187. See also Craigie, *Edinburgh Medical and Surgical Journal*, vol. lvi.



Amongst the consequences of chronic stenosis of the aorta, are: dilatation of this vessel above, and narrowing below the seat of constriction; hypertrophy and dilatation of the left ventricle; and enlargement of the collateral arteries. If dilatation extend to the orifice of the aorta, the valves may become relatively inadequate.

Death may result from rupture of the aorta, cerebral congestion or apoplexy, local inflammation with thrombosis of the aorta, embolism, or from various intercurrent diseases.

The symptoms are inconclusive, owing to the early establishment of a collateral circulation. There is, however, a marked contrast, as to size and force of pulsation, between the mammary, intercostal, and epigastric arteries, engaged in this subsidiary function, and those of the lower limbs. The only acoustic sign noted has been recorded by Blakiston; viz., a postsystolic murmur, loudest at the right second costal cartilage, and transmitted through the aorta.\* But a murmur of this site and rhythm might equally depend upon local roughening of the vessel.

*Aneurism of the coronary arteries* is of rare occurrence. It is, when present, usually associated with atheroma or calcification of the coats of these vessels, and is, therefore, a disease of middle or advanced age. A remarkable example of it in a boy of seven years, has, however, been published by Dr. Gee. In this case, there were three aneurisms; viz., one of the size of a pea at the apex of the heart, another of the same size at the base of the right ventricle close to the origin of one of the coronary arteries, and a third, as large as a horse-bean, in the posterior inter-ventricular sulcus near the base of the heart.†

The disease usually proves fatal by hæmorrhage into the pericardium.

*Aneurism of the valves of the heart* is less rare than that of the coronary arteries. The aortic valves are usually its seat, but it has been met with also in the mitral valves.‡ A case has been

\* Quoted by Walshe (*opus citat.*), whose work may be consulted with great advantage in reference to the literature of this subject. See also the Memoir of Mr. Nixon, *Dublin Journal of Medical and Chemical Science*, vol. vii.

† *St. Bartholomew's Hospital Reports*, vol. vii.

‡ *Vide* three examples by Mr. Prescott Hewett, *London Medical Gazette*, vol. xi. p. 980.

published by Sir Henry Marsh, in which a large pouch, distended with blood, and resting against the anterior segment of the mitral valve, had been formed in one of the aortic valve-segments; it had ruptured into the left ventricle by three rents. The acoustic signs were those of obstruction and inadequacy of aortic valves.\* Crisp refers to two examples preserved in the museum of St. Thomas' Hospital, and in that of Chatham. A case has been also published by Professor Harrison.†

As to the pathology of this affection, Niemeyer thinks it is due to perforation of one of the coats of the valves, with pouching and distention of the other.

*Acute aortitis* is occasionally met with in connexion with atheromatous change of the coats of the vessel. The ascending portion is its usual seat. It is characterized by extreme vascularity and softening of the *intima*, which may lead to rupture of the vessel. During life, it is indicated by palpitation, dyspnoea, and substernal pain, with a feeling of constriction of the chest. A harsh systolic murmur, transmitted to a distant portion of the aorta from roughening of the *intima* at the seat of inflammation, may be also developed, as in the case recorded by Dr. Parkes.‡

*Rupture of the aorta* may result from shock or succussion of the chest. When such an accident occurs, the vessel must have been previously unsound. An example, which was probably of this kind, has been published by Dr. Walshe.§ Death by extravasation may be the immediate result of complete rupture of all the coats; or, the external coat remaining unbroken, death may occur at a later period by its rupture, or a dissecting aneurism may be established.

*Perforation of the aorta* by a penetrating wound, or by a sharp body swallowed and impacted in the oesophagus, is usually fatal within a few minutes.

\* *Proceedings of the Pathological Society of Dublin*, November 28th, 1840.

† *Dublin Journal of Medical Science*, vol. xv., p. 298.

‡ *Medical Times*, February 23rd, 1850.

§ *Opus citat.*, fourth edition, p. 575.



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